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A Dissertation on

ACUTE STROKE DIAGNOSIS AND OUTCOME ASSESSMENT USING NATIONAL INSTITUTES OF HEALTH STROKE SCALE (NIHSS)

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CERTIFICATE

This is to certify that **Dr. P.V.BALAMURUGAN,** Post -Graduate Student (July. 2003 to September 2006) in the Department of Internal Medicine, Kilpauk Medical College, Chennai- 600 010, has done this dissertation on **"ACUTE STROKE DIAGNOSIS AND OUTCOME ASSESSMENT USING NATIONAL INSTITUTES OF HEALTH STROKE SCALE (NIHSS)"** under my guidance and supervision in partial fulfillment of the regulations laid down by the Tamilnadu Dr.M.G.R. Medical University, Chennai, for M.D. (General Medicine), Degree Examination to be held in September 2006.

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ABBREVIATIONS

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ABBREVIATIONS

ABC's	- AIRWAY, BREATHING, CARDIAC STATUS.
ACA	- ANTERIOR CEREBRAL ARTERY
ADL	-ACTIVITIES OF DAILY LIVING
AH	- ACUTE HOSPITAL
AF	- ATRIAL FIBRILLATION
BBB	- BLOOD BRAIN BARRIER
BP	- BLOOD PRESSURE
CVA	- CEREBRO VASCULAR ACCIDENT
DBP	- DIASTOLIC PRESSURE
DHI	- DISCHARGE HOME INDEPENDENT
DHA	- DISCHARGED HOME ASSISTANCE
DM	- DIABETES MELLITUS
GCS	- GLASGOW COMA SCALE
HDL	- HIGH DENSITY LIPOPROTEINS
ICP	- INTRA CRANIAL PRESSURE
IHD	-ISCHEMIC HEART DISEASE
ICA	-INTERNAL CAROTID ARTERY
ICH	-INTRA CEREBRAL HEMORRHAGE
LDL	-LOW DENSITY LIPOPROTEINS

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MCA	-MIDDLE CEREBRAL ARTERY
MMSE	-MINI-MENTAL STATE EXAMINATION
NIHSS	-NATIONAL INSTITUTES OF HEALTH STROKE SCALE
NINDS	-NATIONAL INSTITUTE OF NEUROLOGICAL DISORDERS AND STROKE
OCP	-ORAL CONTRACEPTIVE PILLS
PACS	-PARTIAL ANTERIOR CIRCULATION STROKE
PCA	-POSTERIOR CEREBRAL ARTERY
POCS	-POSTERIOR CIRCULATION STROKE
rtPA	-RECOMBINANT TISSUE PLASMINOGEN ACTIVATOR
RHD	-RHEUMATIC HEART DISEASE
SAH	-SUB ARACHNOID HEMORRHAGE
SHT	-SYSTEMIC HYPERTENSION
SBP	-SYSTOLIC BLOOD PRESSURE
SOL	-SPACE OCCUPYING LESION
TACS	-TOTAL ANTERIOR CIRCULATION STROKE
TIA	-TRANSIENT ISCHEMIC ATTACK

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AIM OF THE STUDY

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AIM OF THE STUDY

Aim of this prospective study is

1) To apply **NATIONAL INSTITUTE OF HEALTH STROKE SCALE (NIHSS)** on patients suspected to have acute stroke and there by to diagnose stroke and to assess outcome of stroke in them, and also

2) To analyze various etiological factors and pattern of clinical presentation with reference to history symptomatology and investigations.

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INTRODUCTION

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INTRODUCTION

DEFINITION:

Acute stroke or Brain attack is defined as focal neurological dysfunction of abrupt onset, which can be attributable to a vascular cause[51].

IMPACT OF STROKE:

The estimated direct and indirect cost of acute stroke in 2005 is estimated to be $56.8 million in the United States alone. More than 7,00, 000 people in the United States have a stroke each year, of which nearly one third represent recurrent events .About 163 000 annual stroke deaths make it the third leading cause of death in the United States[42].

Strokes are even more important because of prolonged disability they cause. The history of world has undoubtedly been altered by stroke. Many important leaders in science, medicine and politics have had their productivity cut permanently or prematurely short by stroke[51]. Between 15% and 30% of stroke survivors become permanently disabled, while 20% remain in institutional care three months after their stroke. The economical and psychological costs of stroke are enormous.

INCIDENCE:

Incidence of stroke has increased considerably in India and other developing countries. The increasing incidence of stroke may be a reflection of

increased longevity, adoption of high fat diet, sedentary life style all made possible by increasing affluence.

CHANGING TREND

Earlier, the clinical examination functioned primarily to catalog a patient's neurological impairments that in turn correlated with the stroke's vascular territory and likely cause. The inferences about the anatomy and etiology guided secondary preventive strategies.

Now despite the advent of modern non-invasive neuroimaging technologies, the clinical examination for stroke is now more important than ever because, therapeutic interventions for patients with acute stroke and sophisticated approaches to prevent recurrent strokes now exist. Appropriate treatment and prevention depend on accurate interpretation of the patient's symptoms and clinical examination findings.

TIME IS BRAIN

Now acute stroke therapy is time dependent. Intravenous recombinant tissue plasminogen activator (rtPA) is almost four times more effective at 1 hour than at 3 hours. The brain can withstand profound ischemia for only limited periods, and the benefits of intravenous tissue plasminogen activator (tPA) lessens as the time from the onset of the patient's symptoms increases[20] .The rapid screening of patients with neurological symptoms begins with

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Prehospital care personnel because the effectiveness of reperfusion strategies for acute ischemic stroke are time dependent. Public education programs have stressed the need to call emergency medical responders for persons experiencing stroke symptoms [11,12]. Arrival to the hospital by emergency medical transport has been associated with more rapid treatment, and there by presumably improved outcomes. Patients, family members, and prehospital care personnel such as emergency medical technicians must recognize the symptoms and signs of strokes to minimize treatment delays. The available data indicate that a physician's assessment of ischemic stroke subtype based on history and physical examinations alone is not reliable. The accuracy of the clinical examination becomes relevant not just for stroke specialists and emergency physicians, but for paramedics, nursing personnel, and emergency medical technicians who may be the first responder. When patients with stroke symptoms arrive at the hospital, a standardized neurological examination scale(like NIHSS) combined with neuroimaging results determine subgroups of patients who might benefit from intravenous thrombolysis versus those who may be at increased risk from thrombolysis-related bleeding [17].

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STROKE DIAGNOSIS –STROKE MIMICS

Diagnosis of stroke is not easy always. Diagnosis is difficult if patient presents with altered level consciousness. A variety of conditions can mimic TIA or stroke. Seizures, neoplasms, infection, intracranial hemorrhage, as well as hypoglycemia and other metabolic abnormalities are among the conditions that can simulate a TIA and stroke [37,38,56]. So keeping these in mind National Institutes of Health stroke Scale (NIHSS) was tested in patients presenting with stroke in ED was found to be useful not only in diagnosing stroke but also to stratify patients so that outcome could be predicted and also to select those patients who will benefit from acute intervention. Among various stroke scales, NIHSS has been studied extensively and its reliability and validity are well documented in scientific literature[33].

So NIHSS was selected for this study and applied on patients suspected to have stroke and its effectiveness in diagnosing stroke and assessing its outcome was studied and confirmed.

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NATIONAL INSTITUTES OF

HEALTH STROKE SCALE (NIHSS)

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NATIONAL INSTITUTES OF HEALTH STROKE SCALE (NIHSS)

1a. ***Level of consciousness***

0 = Alert; keenly responsive

1 = Not alert but arousable by minor stimulation to obey, answer, or respond

2 = Not alert, requires repeated stimulated to attend, or is obtunded and requires strong or painful stimulation to make movements (not stereotyped)

3 = Responds only with reflex motor or autonomic effects or totally unresponsive, flaccid, areflexic

1b. ***Level of consciousness***

Patient is asked the month and age.

0 = Answers both questions correctly

1 = Answers one question correctly

2 = Answers neither question correctly

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1c. ***Level of consciousness***

Patient is asked to open and close eyes and to grip and release nonparetic hand.

0 = Performs both tasks correctly

1 = Performs one task correctly

2 = Performs neither task correctly

2. ***Best gaze***

0 = Normal

1 = Partial gaze palsy, when gaze is abnormal in one or both eyes, but where forced deviation or total gaze paresis is not present

2 = Forced deviation, or total gaze paresis not overcome by oculocephalic maneuver

3. ***Visual***

0 = No visual loss

1 = Partial hemianopia

2 = Complete hemianopia

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3 = Bilateral hemianopia

4. ***Facial palsy***

0 = Normal symmetric movement

1 = Minor paralysis

2 = Partial paralysis

3 = Complete paralysis of one or both sides

5. ***Motor arm***

Scored for right and left arm

0 = No drift, arm holds 90 degrees if sitting (or 45 degrees if supine) for full 10 s

1 = Drift, arm holds 90 degrees if sitting, (or 45degrees if supine) but drifts down

before full 10 s; does not hit bed or other support

2 = Some effort against gravity, arm cannot get to or maintain 90 degrees if

sitting (or 45degrees if supine), drifts down to bed, but has some effort

against gravity

3 = No effort against gravity, arm falls

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4 = No movement

UT = Amputation, joint fusion

6. ***Motor leg***

Scored for right and left leg

0 = No drift, leg holds 30 degrees position for full 5 s

1 = Drift, leg falls by the end for the 5 s period but does not hit bed

2 = some effort against gravity; leg falls to bed by 5 s but has some effort against gravity

3 = No effort against gravity, leg falls to bed immediately

4 = No movement

UT = Amputation, joint fusion

7. ***Limb ataxia***

0 = Absent

1 = Present in one limb

2 = Present in two limbs

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8. ***Sensory***

0 = Normal; no sensory loss

1 = Mild to moderate sensory loss; patient feels pinprick is less sharp or is dull on the affected side, or there is loss of superficial pain with pinprick but patient is aware of being touched

2 = Severe to total sensory loss; patient is not aware of being touched in the face, arm, and leg

9. ***Best language***

0 = No aphasia; normal

1 = Mild to moderate aphasia; some obvious loss of fluency or facility of comprehension, without significant limitation of ideas expressed or form of expression; reduction of speech and/or comprehension makes conversation about provided material difficult or impossible

2 = Severe aphasia; all communication is through fragmentary expression; great need for inference, questioning, and guessing by the listener; range of information that can be exchanged is limited; listener carries burden of

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communication

3 = Mute, global aphasia; no usable speech or auditory comprehension

10. ***Dysarthria***

0 = Normal

1 = Mild to moderate; patient slurs at least some works and at worst can be understood with some difficulty

2 = Severe; patient's speech is so slurred as to be unintelligible in the absence of or out of proportion to any dysphasia, or is mute/anarthric

UT = Intubation or other physical barrier

11**. *Extinction and inattention***

0 = No abnormality

1 = Visual, tactile, auditory, spatial, or personal inattention or extinction to bilateral simultaneous stimulation in one of the sensory modalities

2 = Profound hemi-inattention or hemi-inattention to more than one modality. Does not recognize own hand or orients to only one side of space.

UT - UNTESTABLE

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LITERARY REVIEW

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ACUTE STROKE -LITERARY REVIEW

Diagnosis and treatment of stroke patients require a basic understanding of the anatomy, physiology and pathology of the brain and its blood vessels[51]. .

NORMAL VASCULAR ANATOMY OF BRAIN [51]:

By convention, the carotid artery territories are referred to as the anterior circulation (front of the brain) whereas the vertebral and basilar arteries and their branches are termed the posterior circulation (backof the brain). Each internal carotid artery (ICA) supplies roughly two fifths of the brain by volume, whereas the posterior circulation accounts for approximately one fifth of the total. Despite this much smaller size, it is important to note that the posterior circulation contains the brain stem, a midline strategically crucial structure.

Pathology:

There are two major categories of brain damage in stroke patients,

1. ISCHEMIA: A lack of blood flow depriving brain tissue of needed fuel and oxygen.
2. HEMORRHAGE :The release of blood into brain and extra vascular space within the cranium, which in turn damages the brain by cutting off connecting pathways and by causing localized or generalized injury.

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ISCHEMIA[51]

It can be further subdivided into 1.thrombosis 2.embolism and 3.decreased systemic perfusion.

Thrombosis :

It refers to obstruction of blood flow due to localized occlusive process within one or more blood vessels. The most common vascular pathology is atherosclerosis. They can affect large arteries or atheromatous plaques (microatheromas) can obstruct penetrating arteries. Other less common vascular pathologies are

1. Fibromuscular dysplasia
2. Arteritis (takayasu, giant cell type etc.)
3. Dissection of vessel wall
4. Hemorrhage into plaque

Embolism:

Here material (emboli) formed elsewhere within the vascular system lodges in the distal vessel and blocks the blood flow.

These materials arise proximally, more commonly from the heart, then from major arteries such as aorta, carotid, vertebral arteries and from systemic veins. Cardiac sources include heart valves, endocardium and clot or tumors within the atrial or ventricular cavities.

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Decreased systemic perfusion:

Most common causes are cardiac pump failure (most often due to myocardial infarction or arrhythmias), systemic hypotension, due to blood loss or hypovolemia. Poor perfusion is most critical in border zone or so called *watershed regions* at the periphery of major vascular supply territories.

Damage caused by ischemia by all these three manifestations can lead to temporary or permanent tissue injury. Permanent injury is termed ***cerebral infarction.***

HAEMORRHAGE

It can be subdivided into two subtypes

1. Intracerebral hemorrhage (ICH) and
2. Sub arachnoid hemorrhage (SAH).

These two types; pose different clinical problems and have different management.

Intra-Cerebral Hemorrhage (ICH)

Terms intracerebral or parenchymal hemorrhage refer to bleeding directly into the brain substance. The most common cause is systemic hypertension (SHT), with leakage of blood from small intracranial arterioles damaged by elevated BP.

Other causes include drugs, trauma, vasculopathies, vascular

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Malformations etc. They occur in localized regions and degree of damage depends on the location, rapidity, volume and pressure of the bleeding.

Sub Arachnoid Hemorrhage (SAH):

Here bleeding most often originates from aneurysm or arteriovenous malformation, but bleeding diatheses or trauma can also cause SAH. The blood leaks out of the vascular bed on to the brain surface and spreads quickly via fluid pathway. As the aneurysm releases blood rapidly at SBP, sudden increase in ICP occurs.

CEREBRAL VENOUS THROMBOSIS

Though most brain ischemia is caused by occlusive arterial disease and thromboembolism, brain edema rarely results from thrombosis of dural sinuses and cerebral and cerebellar veins. The causes include infection, pregnancy, peurperium, OCPs, cancer, coagulopathies, etc

RISK FACTORS AND SYSTEMIC ILLNESSES ASSOCIATED WITH STROKE[51]

Extensive information has been accumulated regarding risk factors for the general category of stroke and brain ischemia. Especially regard to SAH, ICH and brain embolism of cardiac origin, ample data exists. The risk factors help identify individuals at risk for stroke in whom modification of life style might reduce the chance of stroke and other CVDs.

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NON-MODIFIABLE RISK FACTORS

Age, race, sex and family history of cardiovascular risk factors/disease are the most important risk factors of stroke.

Age is probably the risk factor best correlated with stroke.

MODIFIABLE RISK FACTORS

Systemic Hypertension (SHT)

After age, systemic hypertension is the risk factor that most significantly correlates with stroke. The degree of elevation of systolic/ diastolic pressure is correlated with the risk of stroke. It plays a role in multiple mechanisms of stroke. It plays a role in athero-degenerative process in large blood vessels, resulting in occlusive and artery-artery embolic strokes. It's also an important factor in rupture of cerebral aneurysms. It is the most common risk factor associated with lacunar strokes.

Cardiac Disease

It is a direct cause of stroke with the heart as a donor source of emboli to the brain. The embolism occurs in the setting of mitral/aortic valve disease, atrial fibrillation (AF)f any cause, prosthetic heart valves, endocarditis,myocardiopathies, akinetic myocardial segments and ventricular aneurysms .AF is the most common cardiac source of brain embolism.

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Diabetes Mellitus

It is associated with an increased risk of ischemic stroke and increased mortality. Hemorrhagic strokes are relatively ***less common*** in diabetics than in non diabetics. Systemic hypertension is more common in diabetics, so some of the effects attributed to DM may be related to SHT.

Smoking

Convincing epidemiological data now strongly relate cigarettes to an increased risk of stroke and intracranial and extra cranial atherosclerosis. The increased stroke risk applies to middle aged men and women but is especially important in young.

Elevated Blood Lipids:

The elevated LDL-cholesterol and low HDL do increase the stroke risk. The risk is primarily seen in patients younger than 55 years. A relationship between ***low cholesterol and ICH*** has been shown in several reports, the mechanism of which is not known.

Transient Ischemic Attacks (TIA's) [39,40,41]

They are an indication that cerebrovascular disease has already been established. In a study of 36 patients with TIAs, 23 (64%) had CT scan positive infarcts.

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Failure to recognize that a TIA has occurred and failure to diagnose and treat a remediable abnormality causing TIA can be a great personal and family tragedy.

Other modifiable causes are OCP use, obesity, lack of exercise, etc.

DIAGNOSIS OF STROKE

Stroke diagnosis is a clinical diagnosis. With the availability of new effective therapies for Acute stroke, early diagnosis now has become more important.

COMMON LOCALIZATION PATTERNS [31]

Neuroanatomic findings can usually be placed in one of seven categories and can be useful in diagnosis. They are

1) **Left hemisphere lesion** (ICA and its MCAand ACA tributaries):

Aphasia, right limb weakness, right limb sensory loss, right visual field defect, poor right conjugate gaze, difficulty in reading, writing and calculating.

2) **Right hemisphere lesion** (in ICA-ACA-MCA distribution) :

Neglect of left visual face, difficulty in drawing and copying, left visual field defect, left limb motor weakness, left limb sensory loss, poor left conjugate gaze, extinction of the two simultaneous left stimulus (visual or tactile)

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The anterior circulation strokes can be total or partial depending upon the site of occlusion and it is termed as Total anterior circulation infarction syndrome (TACS) or Partial anterior circulation infarction syndrome (PACS) [31]

3) **Left PCA lesion** : Right visual field defect, difficulty in reading with retained writing ability, difficulty in naming colours and objects presented visually, normal repetition of spoken language, numbness and sensory loss in the right limbs.

4) **Right PCA lesion: Left** visual field defect often with neglect, left limb weakness and sensory loss.

5) **Vertebrobasilar territory lesion**: Spinning, dizziness, diplopia,weakness or numbness of all four limbs, crossed motor or sensory findings, ataxia, vomiting, headache in the occiput, mastoid or neck, bilateral blindness or dim vision, nystagmus, amnesia It is also called as Posterior circulation infarction syndrome POCS.

6) Pure motor stroke (involves internal capsule or basis pontis): weakness of arm, face and leg on one side of the body without abnormalities of higher cortical functions, sensory or visual dysfunction or reduced alertness.

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7) Pure sensory stroke (involves thalamus): Numbness, or decreased sensibility of face, arm and leg on one side of the body, without weakness, in coordination, visual or higher function abnormalities.

The last two types may be due to lacunar infarction and they are termed as Lacunar infarction syndrome (LACS) - which can be pure motor stroke, pure sensori stroke, sensori-motor stroke, or ataxic hemiparesis.).

IS IT A STROKE?

As no specific clinical stroke diagnostic tool is available so we are dependent on imaging studies for confirmation of acute stroke. To make the clinical situation worse various conditions can mimic a Acute stroke they are termed as stroke mimics [34]. For a new on set focal neurological deficit i.e. stroke / TIA the differential diagnoses are seizure with (post actual (Todd's) paresis ,tumor, migraine ,metabolic encephalopathy (hypo/ hyperglycemia ,hepatic encephalopathy,hypercalcemia etc)CNS infection, old stroke etc[(75)]

In a series, among 821 consecutive patients initially diagnosed with stroke, 13% were finally determined to have other conditions. The most frequent causes of misdiagnosis were unrecognized seizures, confusional states, syncope, toxins, neoplasm's, and subdural hematoma [30,56].

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INITIAL ASSESSMENT AND MANAGEMENT OF STROKE [60]

It consists of

ABC's,

Serum glucose and Non-contrast head CT.

If the CT scan shows

1) Hemorrhage – then appropriate medical or surgical management

2) Tumor / CNS processes - treat as indicated

3) If Scan is Normal or shows hypo dense area consistent with acute Ischemia - consider thrombolysis, aspirin, Maintain BP , hydration, admit patient to appropriate level of care depending on concomitant medical problems.

ROLE OF IMAGING IN ACUTE STROKE [51]

Brain imaging has become an absolutely integral part of evaluation of all patients with Cerebro Vascular Accident (CVA). Both CT and MRI are non invasive and safe, and new generation scanners produce enormous amount of clinically use full information.

Computed Tomography Scan (CT SCAN) [36]

CT remains the most widely used neuro imaging technique for the evaluation of patients with suspected acute ischemic stroke

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It is readily available in most hospitals and can reliably show intra cerebral hemorrhage (ICH).When mechanism is ischemic ,CT may show as a low- density lesion or may initially remain normal. The signs of infarction on CT scans can be subtle when imaged within several hours of the onset of symptoms. Loss of distinction between grey and white matter, obscuring of basal ganglia density ,loss of definition of the insula , and slight hypodensity within infarct zone are often visible in patients with acute brain infarcts.

SAH is not reliably diagnosed by CT as increased density is in the CSF adjacent to the bone and especially if bleed is minor or has occurred days previously. So in these circumstances when SAH is suspected lumbar puncture (LP) may be required to confirm or exclude SAH.

Magnetic resonance imaging **(MRI)** [41,51]

MRI is probably more sensitive than CT in detecting ischemic changes, MRI can also show ICH accurately , especially when echo planar and gradient – echo susceptibility weighted images are performed. The appearance of ICH is complex, dependent on the duration of time since bleed and the choice of MRI imaging. In summary MRI shows hemorrhage less dramatically than CT and it requires care full scrutiny of serial images using different techniques.

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CT SCAN* vs *MRI SCAN IN ACUTE STROKE

With the improvement in MRI, MR has, for the most part replaced CT in most instances. There are however, advantages of CT. The advantages are CT is readily available, less expensive ,interpretation is also easy and much less dependent on technique and filming planes compared to MRI. The scanning time is also less compared to MRI which is important in restless patients. The disadvantages are, CT is not as sensitive as MRI in detecting and imaging acute infarcts, it is not accurate in delineating lesions adjacent to bones and spinal cord strokes , its construction time is more when multiple planes are taken.

The ***2005 ischemic stroke guidelines*** (A Scientific Statement From the Stroke Council of the American Heart Association/American Stroke Association) indicated that additional research was needed to determine the utility of MRI as a substitute for CT among patients with suspected acute stroke because detection of acute intracerebral hemorrhage using MRI had not been fully validated.

COMPLICATIONS OF STROKE [51]

Strokes, like many other serious medical illnesses, can be followed by complications. Complications by itself can cause neurological deterioration

and can be serious and life threatening. The most common complications causing death in these patients are brain edema, cardiac abnormalities, pulmonary embolism, aspiration pneumonitis, UTI, bedsores, phlebothrombosis, falls, etc.

STROKE PREVENTION [51]

It is usually separated into primary prevention (strategies to prevent stroke in patients who have never had stroke) and secondary prevention (strategies to prevent stroke recurrence).

Primary Prevention:

Public should be encouraged to stop smoking, exercise regularly, avoid excessive alcohol intake, avoid becoming over weight and to decrease intake of foods high in fat and cholesterol.

Secondary Prevention:

For secondary prevention, identification of the mechanism of initial stroke is most important. Control of BP is the most important strategy. Other strategies include giving HMG-CoA reductase inhibitors (statins) for patients with non stenosing plaques, surgery/angioplasty for severe carotid stenosis (>70%),anticoagulation for patients with AF who have had

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brain embolism etc.

STROKE ASSESSMENT

NEUROLOGICAL SCALES [5,20,25] AND INDEXES USED IN STROKE

There are many scales for assessing different aspects of stroke. They summarized here,

1)Neurological Impairment Scales

The National Institutes of Health Stroke Scale (NIHSS) and the Canadian Neurological Scale evaluate many of the domains of neurological deficits, including motor, sensory, and visual impairments. In a study comparing the usefulness of the baseline NIHSS, the Canadian Neurological Scale, the Middle Cerebral Artery Neurological Score, and Guy's Prognostic Score, the NIHSS was the best predictor of outcome at 3 months (alive at home, alive in care, or deceased). The NIHSS is reproducible, easy, and quick to perform (10 minutes) and correlates with infarct volume and functional outcome 3 months after stroke.

2)Cognitive Scale

About 15% to 25% of stroke patients develop significant cognitive impairment after the acute ischemic event. To screen for the presence of cognitive changes, scale is the Mini-Mental State Examination

(MMSE)[47]. The MMSE has been widely used to screen for cognitive dysfunction in the stroke population. It is an easily administered 30-item questionnaire that assesses orientation, memory, attention, language, and construction functions. Like other cognitive measures that include tests of arithmetic and language, MMSE scores are highly correlated with educational level. The Neurobehavioral Cognitive Status Examination (NCSE) is another method for assessing severity of dementia.

*3)**Language Scales***

Approximately 30% of stroke survivors have some language dysfunction. Speech and language disorders may interfere with a person's ability to return to a functional independent life. Accurate assessment of the underlying deficits is essential for treatment. Language impairments can best be documented by the use of the American Speech-Language-Hearing Association Functional Assessment of Communication Skills for Adults. This instrument measures adequacy, appropriateness, and promptness of verbal responses. Some measures of language function, such as fluency, naming ability, and comprehension, are also assessed in the NIHSS. Other reliable language-assessment instruments are available, including the Boston Diagnostic Aphasia Examination.

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4)Depression scales

Depression, although common, is perhaps the least-treated sequela of stroke. The prevalence of depression after stroke has been estimated to range from 11% to 68%, a third of which is classified as major depression. Depression can result either from the direct biological effect of brain infarction, such as that associated with left anterior cortex and basal ganglia lesions, or a reaction to the significant losses associated with the stroke. Symptoms of depression can be manifested in cognitive deficits, including difficulty with orientation, memory, language, and distractibility. It is sometimes difficult to distinguish depression from dementia because they share similar symptoms, including disorientation, memory loss, and distractibility. Two assessment scales that reliably screen for symptoms of depression in stroke populations are the CES-D scale and the Geriatric Depression Scale.

5)Basic Activities of Daily Living Scales

(BADL)To determine the extent of disability after stroke, self-care activities and ability to live independently are assessed. The Barthel Index[59] is a measure of severity of disability and the most frequently used stroke outcome measure. It has been repeatedly shown to be a reliable

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and valid measure of BADL. A limitation is that it does not capture significant losses in higher levels of physical functions or activities that are necessary for independence in the home and community. It is responsive to change but has definite ceiling effects in persons with mild stroke. The Functional Independence Measure (FIM) is another widely used disability measure.

6)Instrumental Activities of Daily Living Scales (IADL)

Two assessments are recommended in Post-Stroke Rehabilitation: Clinical Practice Guideline. The Philadelphia Geriatric Center (PGC) Instrumental Activities of Daily Living Scale measures IADL at home and in the community.

NIHSS –LITERARY REVIEW

THOMAS BROTT et al (1) first designed the original scale in 1989 as 15-item neurologic examination stroke scale for use in acute stroke therapy trials for measurement of acute cerebral infarction. It consists of 15 test items. On studying this scale it was found that the most interrater reliable item (pupillary response) had low validity.While less reliable items such as upper or lower extremity motor function were more valid.

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Therefore, the scale was altered by National Institutes of Health (NIH) and is called as NATIONAL INSTITUTES OF HEALTH STROKE SCALE (NIHSS) [17].

The NIH Stroke Scale is an 11-item (expandable to 15 items) with 13 specific tests being performed. This graded neurologic examination stroke scale was used to evaluate the effect of acute cerebral infarction like neurologic outcome and degree of recovery for stoke patients. A trained observer rates the patent's ability to answer questions and perform activities. Ratings for each item are scored with 3 to 5 grades with 0 as normal, and there is an allowance for untestable (UT) items. The examination requires less than 10 minutes to complete. The NIHSS examines for level consciousness, vision and gaze, facial palsy and extremity weakness, limb ataxia, sensory deficit, language, dysarthria and inattention .A patient with a completely normal neurological exam and normal mental status will have an NIHSS of 0. The maximum recordable NIHSS score is 42. However, since acute ischemic stroke causes unilateral paralysis and blindness, the maximum score actually is 31 for a stroke patient with complete hemi paresis, hemianopia, hemi neglect, and aphasia. A patient with only minimal facial or extremity weakness with some loss of sensation would have a

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NIHSS score of 1-2. A patient with a slight alteration in mental status, some loss of vision, slight facial droop, complete hemi paresis, sensory loss on the hemi paretic side, mild aphasia, and slight neglect will have and NIHSS of approximately 19. If these findings are noted as being severe, then the NIHSS would approach 31.Patients with an NIHSS score with score <6,6-14 and >14 are considered to have a mild, moderate severe stroke respectively clinically [3].In a study the median (50% above and below) NIHSS score was 14, consistent with a patient who has mild to moderate stroke physical findings.. By addressing four major areas that are tested by the NIHSS, it is possible to estimate a patient's NIHSS score. These four areas are Cranial nerve/visual, motor, level of consciousness, and language/neglect. By assigning a grade to each area concerning deficit (mild/moderate/severe), and a score of two, four, or eight, depending on the severity of the deficit, it is possible to estimate the NIHSS score. For example, if a patient has a severe deficit in all four areas, then the estimated NIHSS score would be 32. A mild deficit in all four areas would lead to an estimated NIHSS score of eight, and a moderate deficit in all four areas suggests an approximate NIHSS score of 16. The features, which were found to predict functional dependence or death, included older age; complete limb paralysis, depression of conscious level and the combination of hemiplegia and

hemianopia with higher cerebral dysfunction.Hemiparesis uncomplicated by hemianopia or higher cerebral dysfunction predicted a return to functional independence. Thus, NIH Stroke Scale is a critical component of acute stroke assessment.

TRAINING [7]

Minimal amounts of training are required to reliably administer NIHSS, even by non-neurologists, which take less than 10 minutes to complete. A trained person will be able to ,1) Identify and assess neurologic deficits in stroke patients 2) Understand the measurement scale for quantifying neurologic deficits in stroke patients 3) Consistently apply appropriate scores for neurologic deficits in stroke patients 4) Use the scale to assess changes in neurologic deficits in stroke patients over time.

NIHSS IN ACUTE STROKE DIAGNOSIS [4,9]

NIHSS can also be used to diagnose stroke apart from assessing stroke out come and disability. Three items identified 100% of patients with stroke they were facial palsy, motor arm, and dysarthria. An Abbreviated NIH Stroke Scale based on these items had a sensitivity of 100% and a specificity of 92%.Based on a prospective observational cohort study, when performed by a physician, the presence of any of three physical examination finding (facial paresis, arm drift, and abnormal speech) were selected from the National

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Institutes of Health Stroke Scale (NIHSS) as the most useful. These 3 items, selected by statistical recursive partitioning techniques, identified patients with stroke with 100% sensitivity and 88% specificity.

NIHSS AND OTHER STROKE SCALES – A COMPARISON [2,3,4,9,10,13]

In a study, three scales were compared to determine which of several commonly used scales best predicted outcome. The stroke scales compared were the National Institutes of Health Stroke Scale (NIHSS), the Canadian Neurological Scale, and the Middle Cerebral Artery Neurological Score. Outcome at 2, 3, 6, and 12 months was categorized as good (alive at home) or poor (alive in care or dead). Of the 408 patients studied, 373 had confirmed acute stroke and completed follow-up. The three-stroke rating scales each predicted 3-month outcome with an accuracy of 0.79 or greater. The NIHSS provided the most prognostic information. Logistic regression showed that the NIHSS added significantly to the predictive value of all other scores. No score added useful information to the NIHSS. A cut point of 13 on the NIHSS best predicted 3-month outcome.

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THE NIHSS ADVANTAGE

The NIHSS is a reliable tool for rapidly assessing the effects of stroke Analysis showed that increasing NIHSS score was a robust and Independent predictor of discharge to rehabilitation or nursing facilities, roughly doubling for each five-point increment.

The interrater reliability for NIHSS is also good [8.]

RETROSPECTIVE ANALYSIS USING NIHSS [23,24,25]

The NIH Stroke Scale (NIHSS) a stroke impairment scale originally designed for prospective scoring, have been used for the retrospective assessment of initial stroke severity.In addition, it is helpful in determining prognosis. The NIHSS is the most commonly used impairment measure in ischemic stroke treatment trials, and its retrospective application for outcome studies would permit direct comparisons with data from the prospective trials. The NIHSS was found to be both reliable and valid when applied retrospectively in a study of patients enrolled in clinical trials who had been prospectively assessed. A retrospective algorithm developed to apply the NIHSS on the basis of data extracted from patients' medical records in an academic hospital setting also appeared to be reliable and valid

studies have demonstrated that the NIHSS (and other scoring systems) canbe calculated retrospectively if there is adequate documentation of the neurological exam. Retrospective NIHSS scoring with the algorithm is reliable and unbiased even when physical examination elements are missing from the written records.

NIHSS IN ACUTE STROKE MANAGEMENT [6,9,10,17]

Five phase III trials of intravenous rtPA have been reported. Approval of this treatment by the FDA in 1996was based on the results of the National Institute of Neurological Disorders and Stroke (NINDS) rt-PA Stroke Study, in which 624 patients with ischemic stroke were treated with placebo or rtPA (0.9 mg/kg IV, maximum 90 mg) within 3hours of symptom onset with approximately one half treated within 90 minutes. The study was conducted in two parts. In part I, the primary endpoint was neurological improvement at 24 hours as indicated by complete neurological recovery or an improvement of 4 points or more on the NIH Stroke Scale. The likelihood of favorable outcome also was affected by the severity of deficits and the patient's age .Those with mild-to-moderate strokes (NIHSS score <14) and those persons younger than 75 had the greatest possibility fora favorable response to treatment.Although the chancesof a complete or nearly complete

recovery among patientswith severe stroke (NIHSS score >20) improved with treatment, overall success in this group of critically ill patients was low. Patients with major strokes (NIHSS score >22) have a very poor prognosis whether or not they are treated with rtPA.24.The current guidelines is to thrombolyse patients suspected of stroke with NIHSS score not less than 4 and not more than 20. The NIHSS score can predict discharge disposition when thrombolysis is used. There is a significant association of NIHSS scores and the presence and location of a vessel occlusion. With an NIHSS score > or =10, a vessel occlusion will likely be seen on arteriography[54], and with a score > or =12, its location will probably be central. NIHSS scores in basilar, internal carotid, and middle cerebral artery M1 and M2 segment occlusions (central occlusions) were higher than in more peripherally located, nonvisible, or absent occlusions. Patients with ***NIHSS*** scores > or =10 had positive predictive values (PPVs) to show arterial occlusions in 97% of carotid and 96% of vertebrobasilar strokes. With an ***NIHSS*** score of > or =12, PPV to find a central occlusion was 91%. In a multivariate analysis, NIHSS subitems such as "level of consciousness questions," "gaze," "motor leg," and "neglect" were predictors of central occlusions

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PROGNOSIS OF STROKE [2,5,10]

The severity of stroke, based on the findings detected by neurological examination, is a strong indicator of prognosis.The initial ***NIHSS*** score provides important prognostic information. Approximately 60% to 70% of patients with an acute ischemic stroke and a baseline ***NIHSS*** score < 10 will have a favorable outcome after 1 year as compared with only 4% to 16% of those with a score >20. The ***NIHSS*** score can also help identify those patients at greatest risk for intracranial hemorrhage associated with thrombolytic treatment. In the ***NINDS*** trial of rtPA, those with a score of 20 or greater on the ***NIHSS*** had a 17% chance of intracranial hemorrhage, whereas the risk of bleeding was only 3% among those with a score =10. Predicting functional outcome among stroke survivors is more complicated than predicting survival. The results of functional outcome assessments vary depending on when the assessments are performed and how outcome is measured. The NIHSS score not only provides a numerical summary of a patient's neurological impairments that allows monitoring for changes in the extent of deficits, but it also helps determine prognosis and the use of specific therapies. One study found that each additional point on the ***NIHSS***, within 24hours of stroke onset, was associated with a decrease in the likelihood of an excellent outcome at 7 days by 24%.As described above, the

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NIHSS predicts a patient's prognosis. Less than 20% of untreated patients with an NIHSS score of more than 15 at baseline recover to the point of having little or no disability. Approximate point estimates predicting outcome at 3 months are based on NIHSS scores obtained within the first 24 hours of ischemic stroke. Thus of the several reliable and well-validated scoring systems that have been developed the ***National Institutes of Health Stroke Scale (NIHSS)*** stands out and will be used widely.

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PATIENTS AND METHODS

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PATIENTS AND METHODS

PLACE OF STUDY

This study was conducted in medical wards' and intensive care unit of .

Government Kilpauk Medical Hospital, Chennai-10.

PERIOD OF STUDY

From April 2005 to January 2006.

DESIGN

Observational prospective study using National Institute of Health Stroke Scale (NIHSS) to diagnose (if NIHSS > 3) and assess outcome of Acute stroke and also to analyze various etiological factors and pattern of clinical presentation of Acute stroke with reference to history, symptomatology and investigations.

METHODOLOGY

A. Subject selection

1. Inclusion criteria

a. Patients diagnosed to have stroke using NIHSS (score >3) plus patients should at least have one of the following clinical feature like facial paresis, leg paresis, arm paresis and aphasia

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b. Symptoms of stroke not lasting more than seven days

2. Exclusion criteria

a. Stroke symptoms lasting more than seven days.

b. Past history of stroke.

All the patients diagnosed to have stroke were thoroughly examined and evaluated in detail as per the proforma prepared for this study. Elaborate history, mode of presentation, past history of TIA's and symptoms were sought and recorded. Symptoms of stroke like gait/language disturbances, headcahe, vomiting, convulsions etc were noted. Similarly, signs of stroke like hemi paresis, facial paresis, hemianopia, altered level of consciousness, diplopia, sensory deficits were recorded Risk factors and systemic illnesses associated with stroke were noted for each patient. NIHSS was applied on all patients suspected to have stroke, patients were selected, and two scores for each patient, one on admission and another at the time of discharge/referral/death were obtained. All patients were subjected to CT-SCAN BRAIN and the radiologist report compared with NIHSS score.

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OBSERVATIONS, ANALYSIS AND DISCUSSION

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OBSERVATIONS

Hundred patients were studied. Their age ranged from 30-84 years (mean 57 yrs). Sixty one of them were males (61%) and 21 females (39%). 66 of them were hypertensives (66%), while 23 had Diabetes and 22 were smokers. Young stroke (age < 40yrs) was noted in 10 patients (10%).

Of the two NIHSS score recorded for each patient the base line score ranged from (3-30) while the discharge score ranged from (0-10).

Through analysis of all the findings and parameters recorded from these patients were don

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TABLE – 1

SEX WISE DISTRIBUTION OF ACUTE STROKE

S.No.	Sex	Number	Percentage
1.	Male	69	69%
2.	Female	39	31%

ANALYSIS AND DISCUSSION

1) Men have a greater frequency of stroke than a woman has, but because life expectancy is higher in women, women often out number men in many stroke studies
2) During the pre menopausal years, women have fewer stroke than men but the incidence levels off after 60 years. ***(Sacco RL, Risk factors , outcomes, and stroke sub types for Ischemic stroke . NEUROLOGY 1997; 47; 1420-1428.)***

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TABLE – 2

AGE GROUP WISE DISTRIBUTION OF ACUTE STROKE

AGE GROUP	MALES	FEMALES	TOTAL	PERCENTAGE
<40	7	3	10	10%
41-50	17	7	24	24%
51-60	16	10	26	26%
61-70	15	17	32	32%
71-80	6	0	6	6%
81-90	0	2	2	2%

ANALYSIS AND DISCUSSION

1) Age is probably the risk factor best correlated with stroke. As the age increases, an exponential increase occurs in the frequency of stroke.

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2) In this study the highest incidence was found in 61-70 age groups. The majority of ischemic strokes occur in persons older than 65 years**.**

(Sacco RL, Risk factors, outcomes, and stroke sub types for Ischemic stroke. Neurology 1997; 47; 1420-1428.)

3) The Framingham study showed that as the person ages his risk of stroke increases, with the incidence rates per 1000 increasing from 22% to 32% to 83% in the age groups 45-54, 55-64, 65-74 years respectively

4) The incidence of SAH also increases with age**. (Teunissen et al, Risk factors for SAH.A systematic review. STROKE 1996:27; 544-549)**

5) Stroke in persons < 40 yrs (YOUNG ***STROKE***) was found in 10 persons (10%). Causes of young stroke are heterogeneous than in older patients. The differential diagnoses include many genetic, congenital, metabolic, drugs, migraine, trauma, hematologic causes and systemic disorders, which are rare in mature adult population. The cause also varies with geography, socioeconomic factors and environmental factors. Hemorrhagic strokes including SAH are more common in young. Occlusions of dural venous sinuses are more common. Brain and

vascular location of lesions are somewhat different in the young. Cerebral infarcts tend to be more limited to deep regions of the hemispheres especially the striato-capsular region .Vascular occlusive lesions are more often intra cranial, affecting especially supraclinoid internal carotid artery, proximal MCA and basilar artery. Because of the absence of diffuse vascular disease and good collateral circulation over the convexities cortical territory of the MCA is spared. Vascular malformations are more often peri-ventricular or intra ventricular than in adults. In young, TIA's are unusual. While seizures, brain edema and ICP more common. Aphasias are more often non-fluent.

Prognosis of young stroke is better than in adults. Developing brain shows, more plasticity and undamaged areas can frequently assume the functions of damaged regions.

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TABLE-3

Activity at onset in subtypes of stroke

ACTIVITY AT ONSET	*THROMBOSIS (%)*	*EMBOLUS (%)*	*HEMORRHAGE (%)*	*LACUNAR (%)*	*NORMAL CT-SCAN (%)*
SLEEP/ ON ARISING	*36*		*15*	*40*	*45*
ADL	*56*	*100*	*85*	*55*	*55*
UNKNOWN	*8*			*5*	

ANALYSIS AND DISCUSSION

1) In this study stroke commonly (78%) occurred when the patient was involved in Activities of Daily Life (ADL). While 56% of thrombotic strokes occurred when the patient was active, 85 % (6/7) of the hemorrhagic stroke occurred when the patient was active.

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2) Traditional teaching states most of the thrombotic strokes occur when the circulation is least active /sluggish (sleep/arising) while hemorrhage and embolism occur when the circulation is more active / BP rises. However, newer data suggest most thrombotic strokes occur between 10 AM and noon and significant number of strokes occur at night. **(Marler J et al.Morning increase in onset of ischemic, stroke, STROKE 1989;20;473-476)**

3) How ever it is unusual for a thrombotic stroke /lacune to develop during vigorous physical activity /coition.

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TABLE – 4

TYPE OF STROKE FROM CT SCAN

S.No.	TYPE OF STROKE	NUMBER	PERCENTAGE
1.	ISCHEMIC /THROMBOTIC	73	76%
2.	HEMORRHAGIC	7	7.2%
3.	LACUNAR	11	11.4%
4.	EMBOLIC	1	1%
5.	NORMAL	4	4.2%

ANALYSIS AND DISCUSSION

1) In this study 76% (73/96) of the stroke were of ischemic type i.e showing infarcts in CT SCAN Brain.11.4% (11/96) of the total strokes were of lacunar type while 7.2% (7/96) .

2) Of the strokes 7(7.2%) were hemorrhagic (ICH). 1% (1/96) of the total stroke was suspected to be of embolic type.

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3) An analysis of data from large stroke studies and registries show that approximately 80% of all strokes are ischemic while 20% are hemorrhagic. *(Foulkes et al. The Stroke Data Bank: designs , methods, and base line characteristics. Stroke 1988; 19; 547-554)*

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TABLE -5

TYPE OF STROKE AND SEX DIFFERENCE

TYPE OF STROKE	MALE	FEMALE	TOTAL
THROMBOTIC	47	26	73
HEMORRHAGIC	5	2	7
LACUNAR	5	6	11
EMBOLIC	0	1	1
NORMAL	2	3	5

ANALYSIS AND DISCUSSION

1) In this study no significant difference between sexes was found with respect to type of stroke except for that, lacunar strokes were more common in females (19%) than in males (7%).

2) Hemorrhagic strokes were common in males (7%) than females (6%).

3) In this series only one embolic stroke was diagnosed in a female patient who was a known Rheumatic Heart Disease patient.

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TABLE – 6

SYSTEMIC ILLNESSES/ RISK FACTORS IN ACUTE STROKE

SYS.ILLNESS/ RISK FACTORS	NUMBER	PERCENTAGE
SHT	66	66%
DM	23	23%
SMOKING	22	22%
ALCOHOL DEPENDENCE	9	9%
H/O TIA	2	2%
IHD	2	2%
RHD	1	1%
HIGH CHOLESTEROL	4	4%
FAMILY HISTORY	2	2%
NO RISK FACTORS	24	24%

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ANALYSIS AND DISCUSSION

1) The hypertensives accounted for nearly 66 % of patients. 23% of the patients were diabetics while, 22% were smokers.

2) With the available data, can be noted that 24% of patients did not have any risk factors for acute stroke.

3) Multiple risk factors (=/> 2) were found in 38 patients and two third of them were males.

4) Family history of stroke was noted in two patients.

-

TABLE – 7

SYMTOMATOLOGY IN ACUTE STROKE

SYMPTOMS	NUMBER	PERCENTAGE
ALTERED LEVEL OF CONSCIOUSNESS	68	68%
SPEECH DISTURBANCE	79	79
GAIT DISTURBANCE	92	92
HEADACHE	8	8
VOMITING	3	3
CONVULSIONS	3	3
VERTIGO/GIDDINESS	4	4

ANALYSIS AND DISCUSSION

1) Among the various symptoms with which the patient presented gait disturbance (92%) was the most common symptom while speech deficit and altered level of conscious ness was found in 79% and 68 % of cases respectively.

2) While loss of conscious ness is rare in ischemic strokes, its presence is an important sign of brain edema or increased ICP or lesions

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of Brain stem ARAS or bilateral cerebral hemispheres ***(Young.GB.et al,Coma and Impairment Consciousness ; A Clinical Perspective McGraw-Hill-1998).***

3) Seizures are in early part of stroke and their presence argues for embolic stroke or ICH. Vomiting is rare with anterior circulation strokes but vomiting in anterior circulation stroke usually equal ICH. Decreased in level of consciousness is unusual in lacunar stroke.

(Louis .R.Caplan, Caplan's stroke; A Clinical approach, Third Edition)

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TABLE – 8

CLINICAL SIGNS IN ACUTE STROKE

SIGNS	NUMBER	%
HEMI PARESIS	80	80
FACIAL PARESIS	78	78
APHASIA	65	65
DYSARTHRIA	14	14
HEMIANOPIA	1	1
DIPLOPIA	1	1
MONOPAERESIS	1	1
SENSORY DEFICIT	1	1

ANALYSIS AND DISCUSSION

1) The most common clinical sign recorded was hemi paresis

Hemiplegia (80%), followed by facial paresis (78%) and aphasia (65%). Facio- brachial monoplegia was noted in one patient .similarly sensory deficit could be made out objectively in only one patient.

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2) For patients with right hemisphere lesions with dysfunction of left limbs or visual field it is important to test visuo spatial functions and also for left side neglect, which is manifested by omitting words ,phrases ,or people on the side of the page**. (Heir et al.Behavioural deficits after Right hemisphere stroke Neurology 1983; 33; 337-344)**

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TABLE – 9

LOCATION OF INFARCTION

S.NO	LEFT HEMISPHERE	RIGHT HEMISPHERE	BI LATERAL	BRAIN STEM
1	36	53	1	1

ANALYSIS AND DISCUSSION

1) In this study right hemisphere, lesions (58%) were more common than left hemisphere lesions (39%). Bilateral acute infarct and brain stem infarct were found in one CT-SCAN each.

2) CT –SCAN was normal in 4 patients. When mechanism is ischemic, CT may show as a low- density lesion or may initially remain normal. The signs of infarction on CT scans can be subtle when imaged within several hours the onset of symptoms. [36, 51]

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TABLE 10

LOCATION OF INFARCT AND CLINICAL SIGNS

	LEFT HEMISPHERE	%	RIGHT HEMISPHERE	%
FACIAL PARESIS	32	89	46	86
HEMI PARESIS	32	89	48	90
APHASIA	30	83	33	62
DYSARTHRIA	2	5	14	26
HEMIANOPIA / NEGLECT	0	0	4	4
DIPLOPIA	1	2	0	0
MONOPARESIS	1	2	0	0
SENSORY DEFICIT	0	0	1	1

ANALYSIS AND DISCUSSION

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1) In this study for both right and left hemisphere lesions hemi paresis was the commonest clinical sign, followed by facial paresis and aphasia.

2) Aphasia was noted in 90% of left hemisphere stroke but only in 62% of right hemisphere stroke. Patient may have aphasia alone ,which places the lesion in left hemisphere anterior circulation as no other pattern includes aphasia.

3) Sudden abnormality in behaviour is noted in caudate nucleus ,thalamic and frontal lobe lesions**.(Mendez et al,neurobehaviour change associated with caudate nucleus .Neurology 1989; 39;349-354)** and **(Caplan LR et al ,caudate infarcts Arch Neurology 1990;47;133-143.)**

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TABLE 12

NIHSS AND AGE GROUP

AGE GROUP ↓	NIHSS <6 MILD	NIHSS 6-14 MODERATE	NIHSS >14 SEVERE
< /= 40	3	5	2
41-50	8	12	4
51-60	6	13	7
61-70	3	22	7
71-80	0	5	1
81-90	0	2	0
MALE	11	41	9
FEMALE	9	18	12

ANALYSIS AND DISCUSSION

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1) In this study the patients diagnosed to have stroke using NATIONAL INSTITUTES OF HEALTH STROKE SCALE (NIHSS) were further divided into mild , moderate and severe stroke based on baseline NIHSS SCORE. Patients with score less than six had mild stroke while those with score 6-14 and >14 were said to have moderate and severe stroke respectively.

2) 61% of patients had moderate stroke while only 18% and 21% of patients had mild and severe strokes .This similar trend was found in both sexes.

Analyzing NIHSS score and age it was found that as the age increases the percentage of patients having moderate to severe stroke increases. For example while only 66% of persons had moderate to severe stroke in 41-50 age group 90% in 61-70 had moderate – severe stroke

2) While only 10% of males had severe stroke 30% of females had severe stroke. Over all severe strokes were common in females.

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TABLE 13

STROKE SEVERITY AND DURATION OF HOSPITAL STAY.

DURATION OF HOSPITAL STAY ↓	NIHSS SCORE <6	NIHSS SCORE 6-14	NIHSS SCORE >14	TOTAL
0-5 DAYS	9	30	11	50
6-10 DAYS	7	24	9	40
10-15 DAYS	4	2	1	7
15-20 DAYS	0	2	0	2
>20 DAYS	0	1	0	1
TOTAL	20	59	21	100

ANALYSIS AND DISCUSSION

1) The average duration of hospital stay was 4-6 days following an episode of Acute stroke. The shortest duration was 2 days while longest duration was 27 days
2) This study did not show any influence of base line NIHSS score over the duration of hospital stay. Usually (50%) patients were discharged within 6 days. 40% got discharged before 10 days.

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TABLE 14

ADMISSION NIHSS SCORE AND ACUTE STROKE OUTCOME

DISCHARGE STATUS/ OUTCOME ↓	NIHSS SCORE <6	NIHSS SCORE 6-14	NIHSS SCORE >14	TOTAL
DISCHARGE HOME INDEPENDENT	17	34	3	52
DISCHARGE HOME ASSISTANCE	2	24	11	37
ACUTE HOSPITAL	1	1	1	3
EXPIRED	0	0	8	8
TOTAL	20	59	21	100

ANALYSIS AND DISCUSSION

1) Of the 89 patients who got discharged 52% were home independent while 37% of patients needed some form of assistance at home after discharge for their daily activities.

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2) Of the 21 patients who had severe stroke (NIHSS >14) , 8 patients (38%) died in the hospital itself. While no patient with mild and moderate stroke died. **(Adams HP Jr, et al. Baseline NIH stroke scale score strongly predicts outcome after stroke: a report of the Trial of Org 10172 in Acute Stroke Treatment (TOAST). Neurology. 1999; 53:126–131.)**

3) About 80% of patients with an NIHSS < 12- 14 will have a good or excellent outcome, whereas only 20% of patients with an NIHSS > 20-26 will have this similar good or excellent outcome

4) Three patients were referred as acute hospital to higher specialties for further management as CT –SCAN showed meningioma, multiple granulomas and frontal SOL in these patients.

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TABLE 15

MODE OF ONSET OF STROKE

ACUTE STROKE –MODE OF ON SET AND COURSE

MODES OF ONSET	NUMBER
SUDDEN WITH MAXIMAL DEFICIT	89
PROGRESSIVE	5
REMISSION	5
FLUCTUATING	1
STEP WISE /STUTTER	0

ANALYSIS AND DISCUSSION

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1) In this study the commonest mode of onset was, sudden with maximal deficit.the modes of presentatation of stroke were progressive (5%), remission (5%) and fluctuating type (1%).
2) The early course gives important information about the stroke mechanism. especially constructing a " course of illness graph" with temporal pattern of findings helps to predict stroke mechanisms.
3) If improvement is seen shortly after onset of the deficit, it argues strongly against ICH, it is most compatible with an embolic mechanism.
4) The gradual development of a progressive focal deficit, accompanied by gradually developing ICP symptoms suggests ICH.
5) If the onset is abrupt and maximal the two stroke highest probability are thrombosis and embolism.
6) Sudden, maximal onset deficits in patients with large artery occlusions are presumed to be caused by artery-to-artery embolism from the donor site of thrombosis to a recipient intracranial artery.

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CONCLUSION

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CONCLUSION

1) The incidence of Acute stroke / Brain attack like coronary artery disease (CAD) is increasing especially in younger individuals (<50 yrs).

2) Stroke is more common in males (61%) compared to females (39%). Similarly, multiple risk factors were also common in males than females. Sixteen out of the total 39 females did not have any risk factors.

3) Females especially those over 60 yrs of age had more severe strokes (NIHSS > 14) compared to their male counterparts of same age group and NIHSS score. Similarly, death was also common in females than males

4) Systemic hypertension (66%) was the most common risk factor/systemic illness associated with stroke followed by DM (23%) and smoking (22%).

5) Gait disturbance is the most common symptom followed by speech disturbance and altered level consciousness in that order.

6)Hemi paresis was the most common clinical sign followed by facial paresis and aphasia and dysarthria.

7) NIHSS is helpful in identifying persons with stroke from those suspected to have stroke (with a sensitivity of 91%)

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8) NIHSS is use full in patient assessment and stratification for further course of management. Among those patients diagnosed to have stroke most (79%) had mild to moderate stroke (NIHSS score = 0-14) compared to 21% who had severe stroke (NIHSS score>14).

9) The NIHSS score on the day of admission predicts the outcome of stroke, volume of infarct in CT – SCAN and thus prognosis of stroke. All those patients who died had their base line NIHSS score more than 24.

10) The NIHSS score on the day of admission(base line score) is useful in selecting patients for whom early intervention (thrombolysis) will be beneficial those (with NIHSS between 4 and 20)

11) Most of the patients with stroke recover satisfactorily with conventional treatment and NIHSS score at the time of discharge reflects these changes.

12) Early recognition of risk factors and specifically treating them can lead to prevention stroke and its permanent disability.

13) Medical and paramedical staffs can be trained to administer NIHSS for early recognition of stroke and effective treatment.

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GRAPHICAL PRESENTATION

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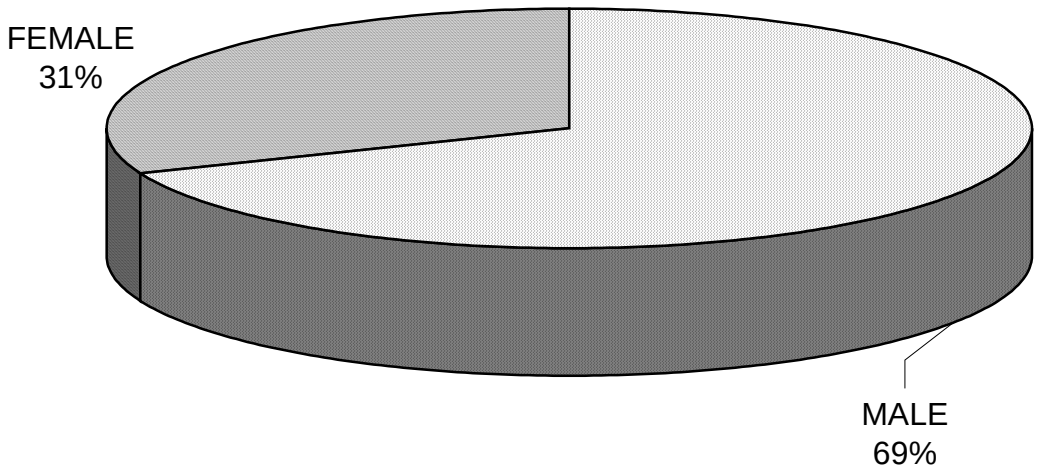
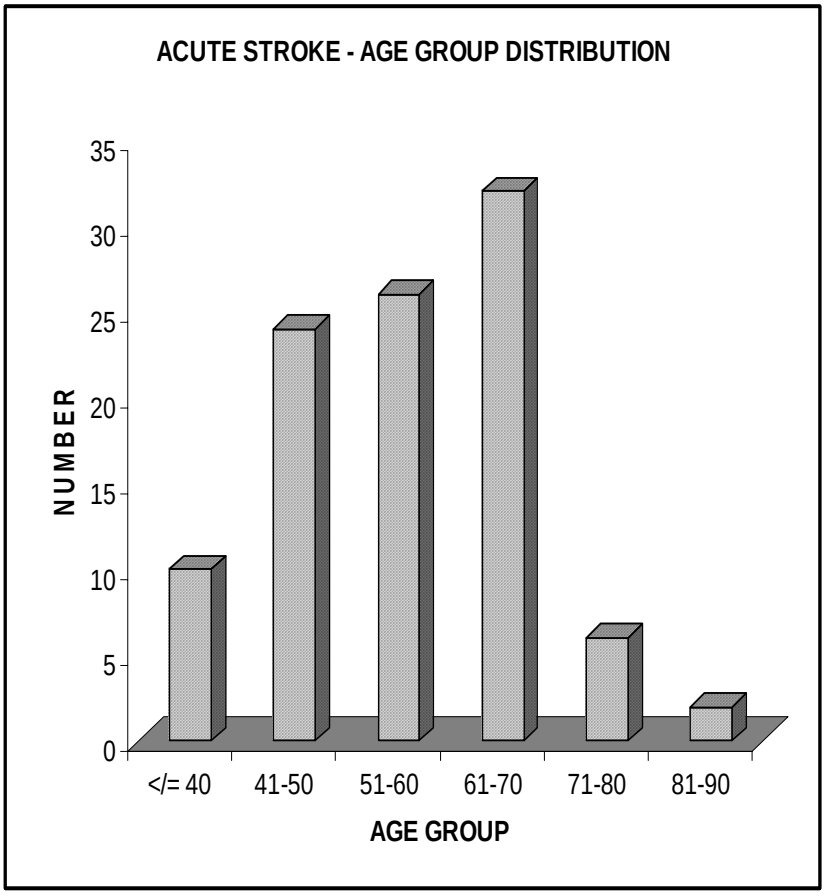
ACUTE STROKE - SEX INCIDENCE



-


ACUTE STROKE - AGE GROUP DISTRIBUTION
NUMBER
35
30
25
20
15
10
5
0
</= 40
41-50
51-60
61-70
71-80
81-90
AGE GROUP

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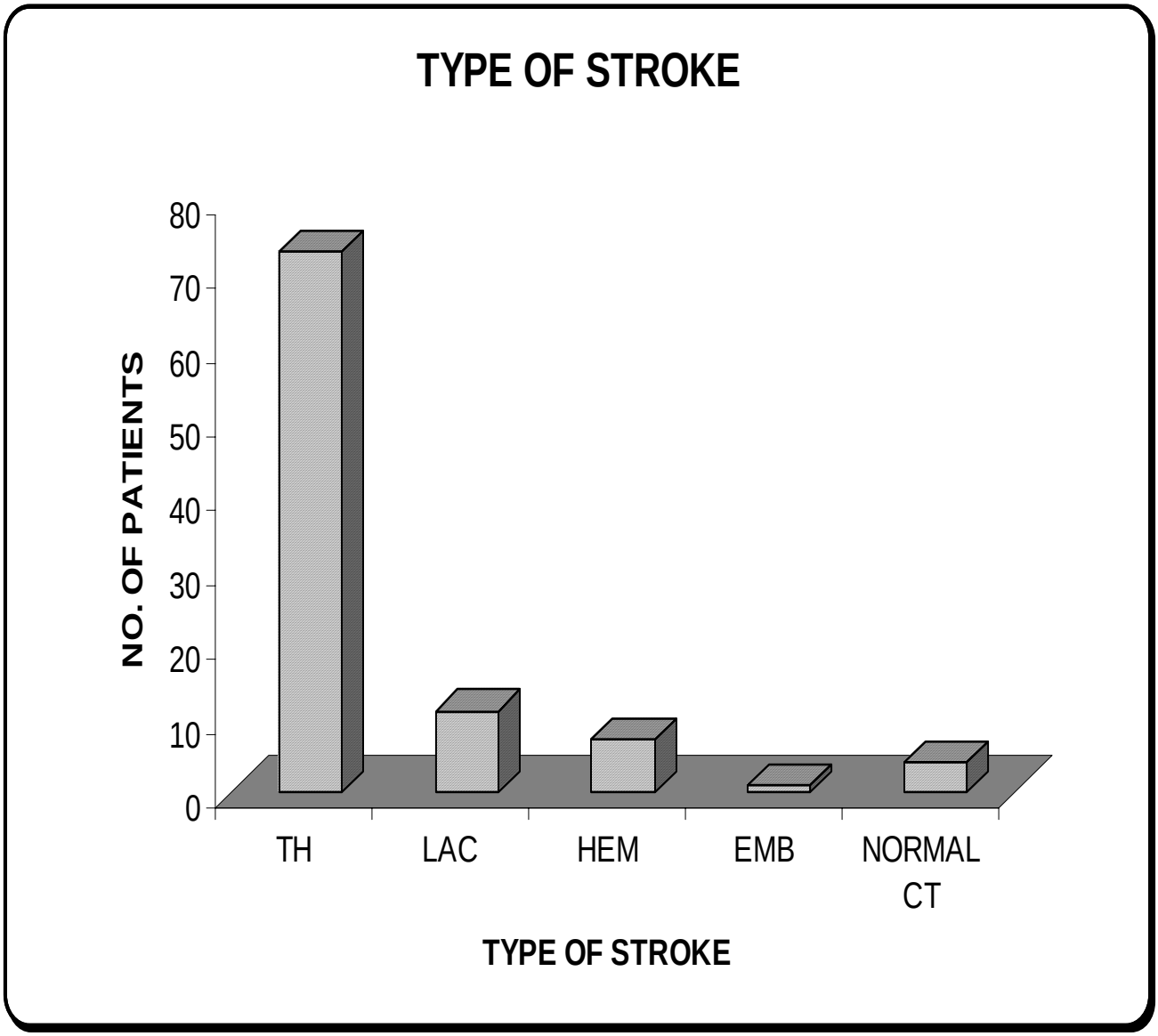
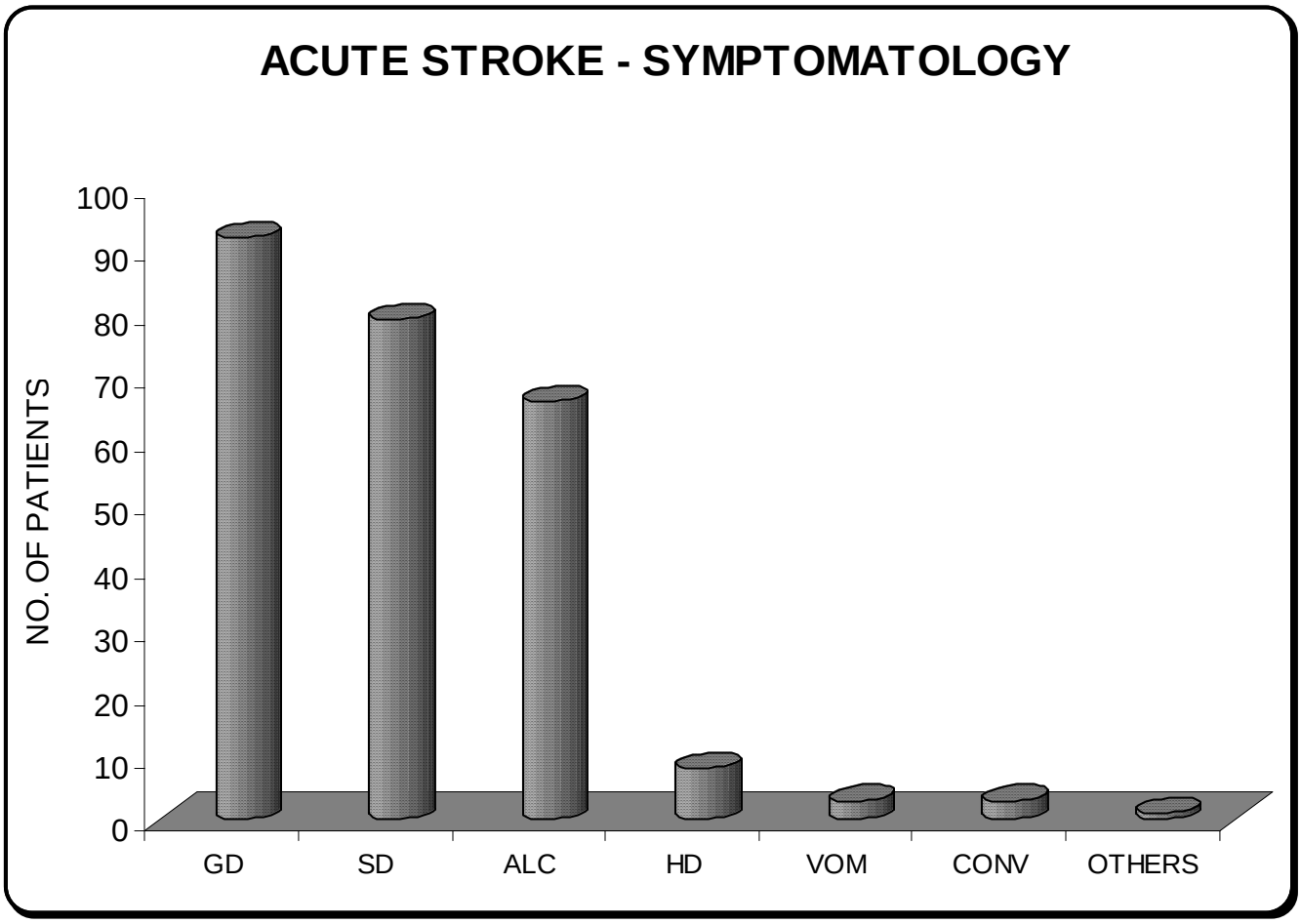


TH **T*HROMBOTIC STROKE.***
LAC ***LACUNAR STROKE.***
HEM ***HEMORRHAGIC STROKE.***
EMB ***EMBOLIC STROKE.***

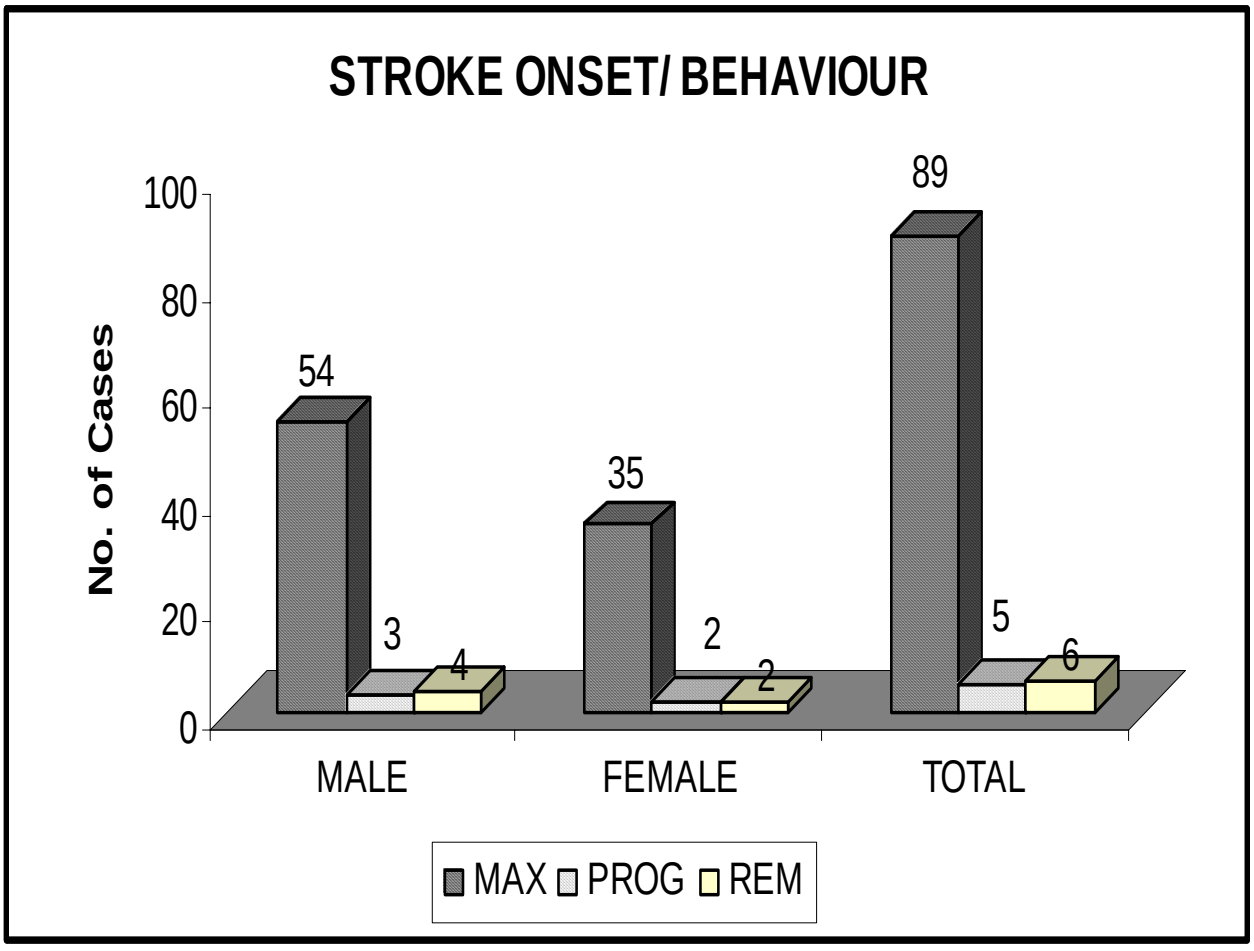
-



GD - ***GAIT DISTURBANCES***
SD - ***SPEECH DEFICITS***
ALC - ***ALTERED LEVEL OF CONSCIOUSNESS,***

HD ***- HEAD ACHE***
VOM ***-VOMITING***
CONV ***- CONVULSIONS***

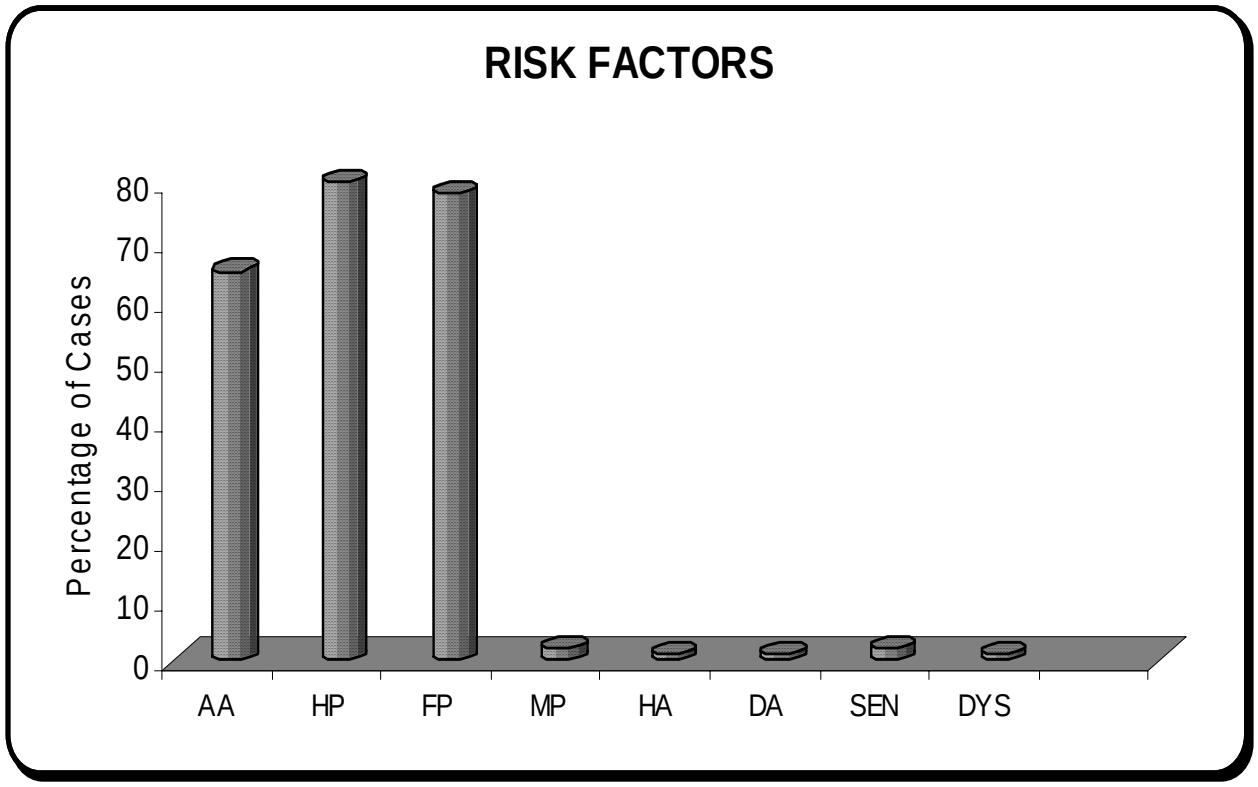
-



MAX – SUDDEN ONSET WITH MAXIMAL DEFICIT AT TIME OF ONSET

PROG – PROGRESSIVE

REM -REMISSION



HP – ***HEMIPARESIS / HEMIPLEGIA***

AA - **APHASIA**

FP – ***FACIAL PARESIS***

DYS – ***DYSARTHRIA***

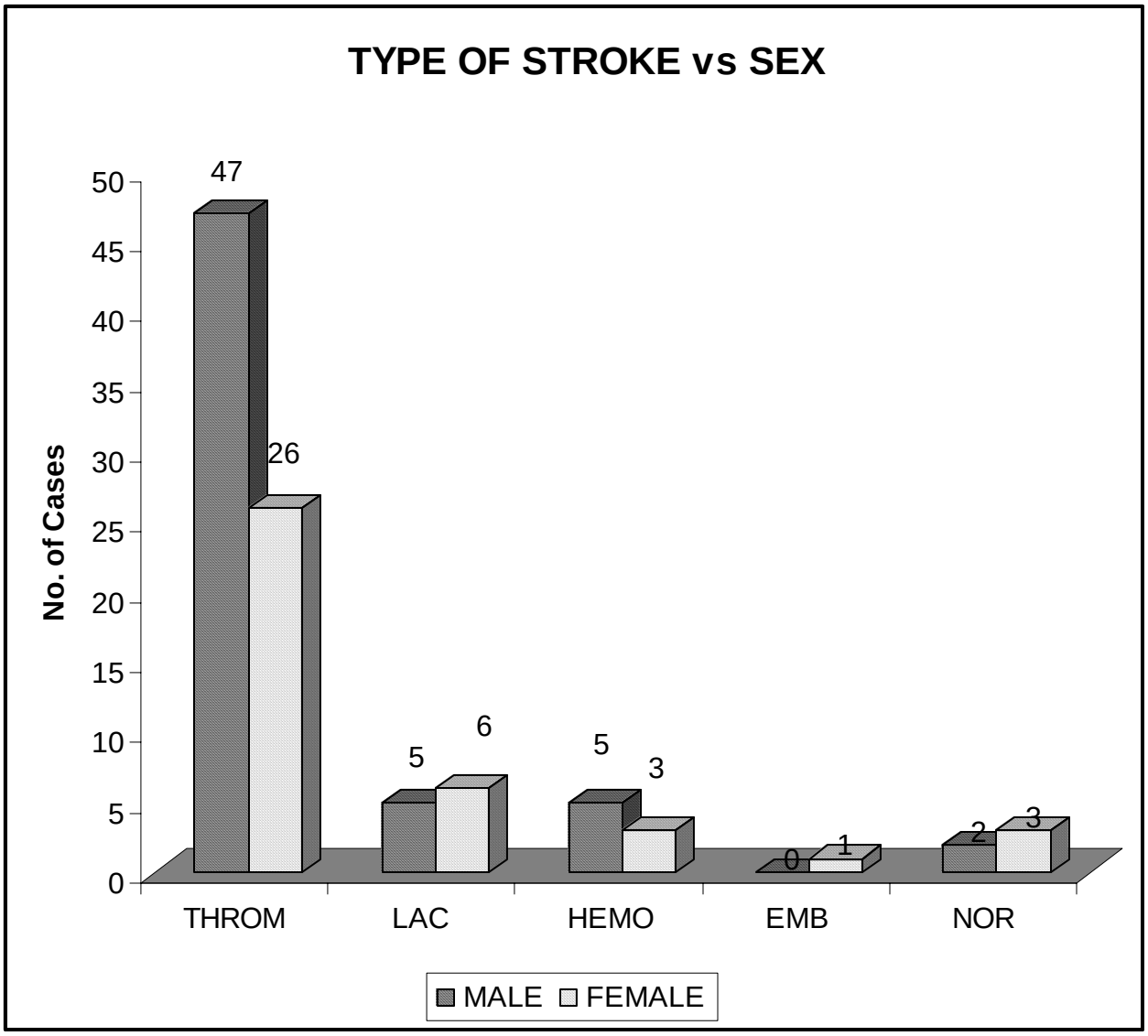
MP – ***MONOPARESIS***

SEN- ***SENSORY DEFICITS***

HA - ***HEMI ANOPIA***

DA - ***DIPLOPIA***

-



TH – *THROMBOTIC STROKE.*

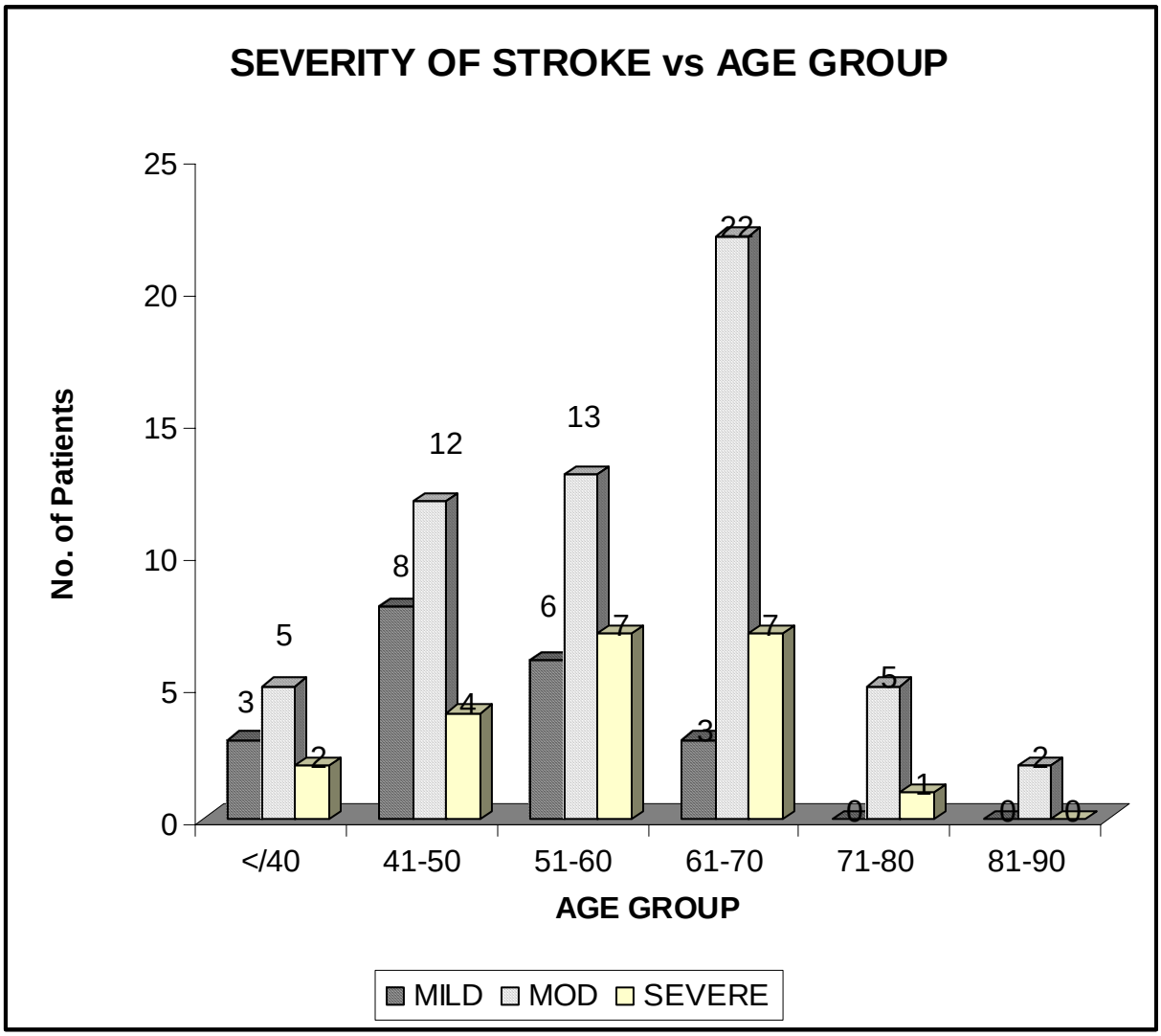
LAC- *LACUNAR STROKE.*

HEMO- *HEMORRHAGIC STROKE*.

EMB- *EMBOLIC STROKE.*

NOR -NORMAL CT -SCAN

-

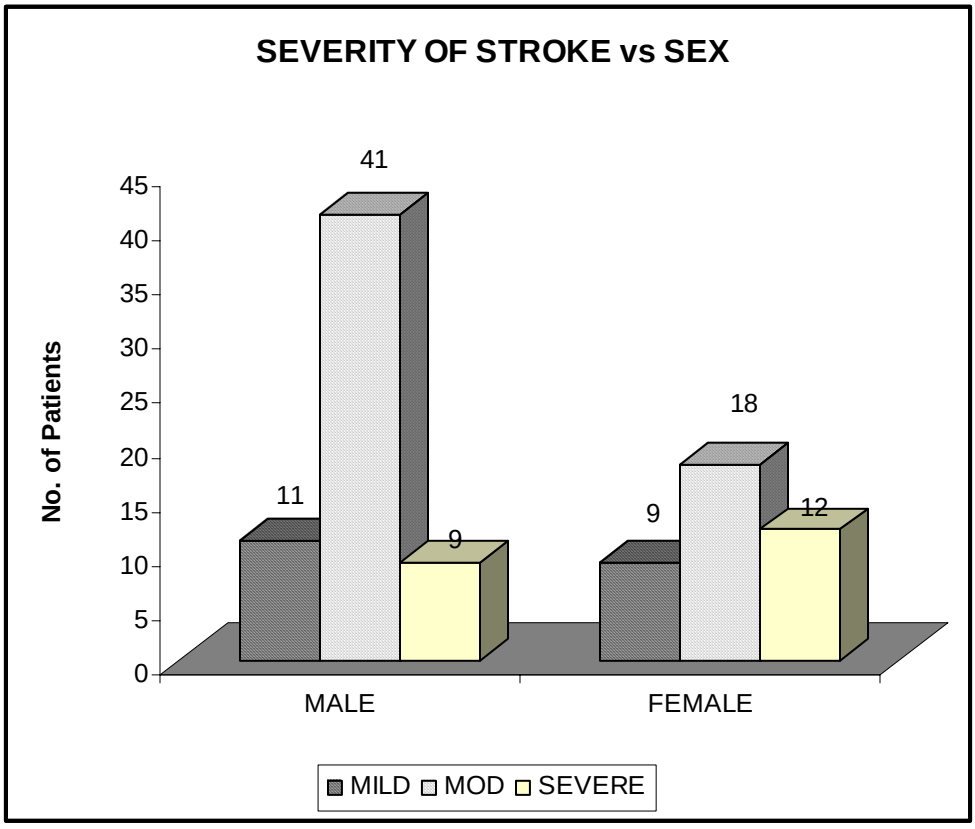


MILD STROKE – NIHSS SCORE <6

MODERATE STROKE –NIHSS SCORE 6-14

SEVERE STROKE – NIHSS SCORE >14

-

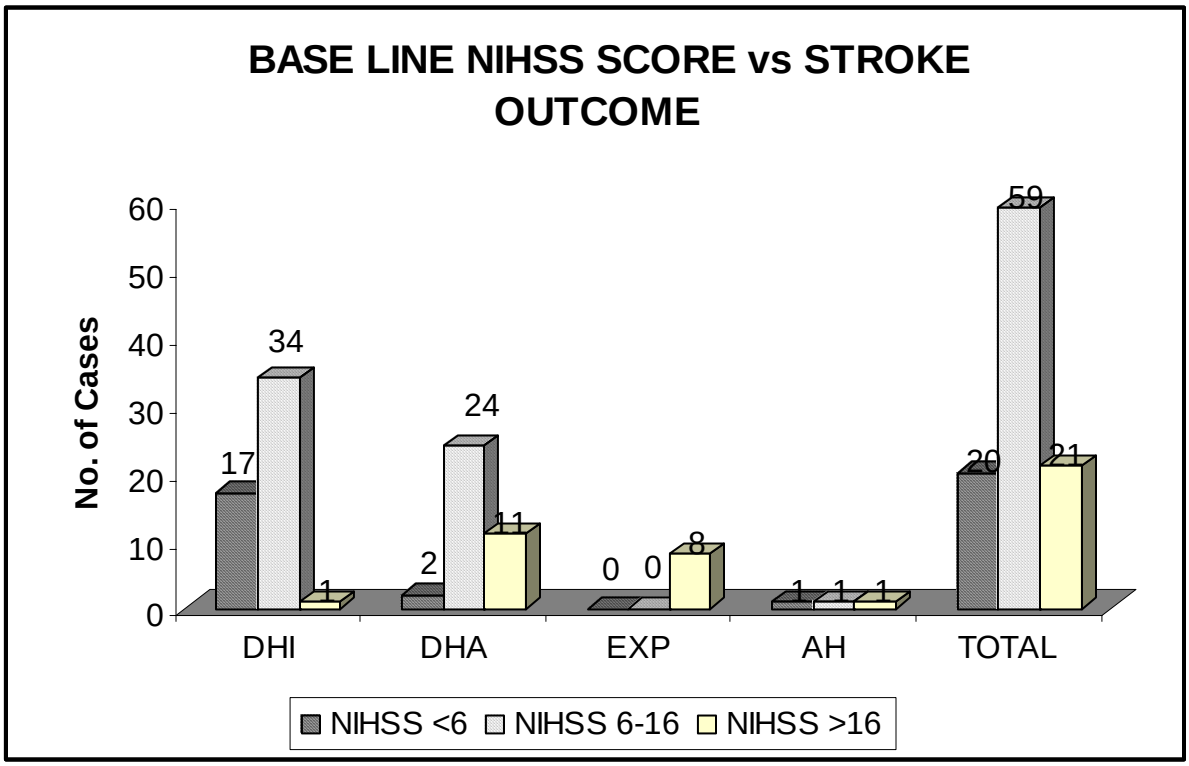


MILD STROKE – NIHSS SCORE <6

MODERATE STROKE –NIHSS SCORE 6-14

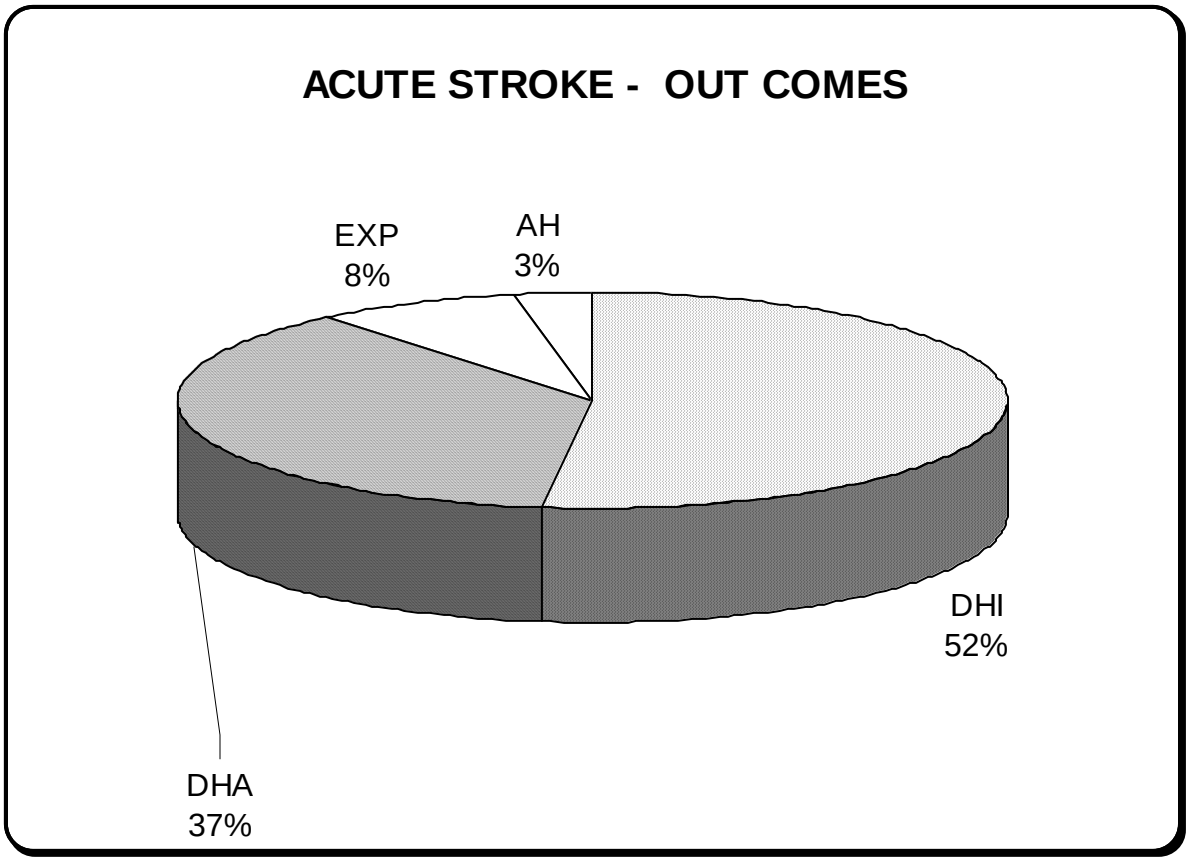
SEVERE STROKE – NIHSS SCORE >14

-



DHI – ***DISCHARGE WITH HOME INDEPENDENCE***
DHA - ***DISCHARGE WITH HOME ASSISTANCE***
AH – ***ACUTE HOSPITAL***
EXP - ***EXPIRED***

-



DHI – *DISCHARGE WITH HOME INDEPENDENCE*
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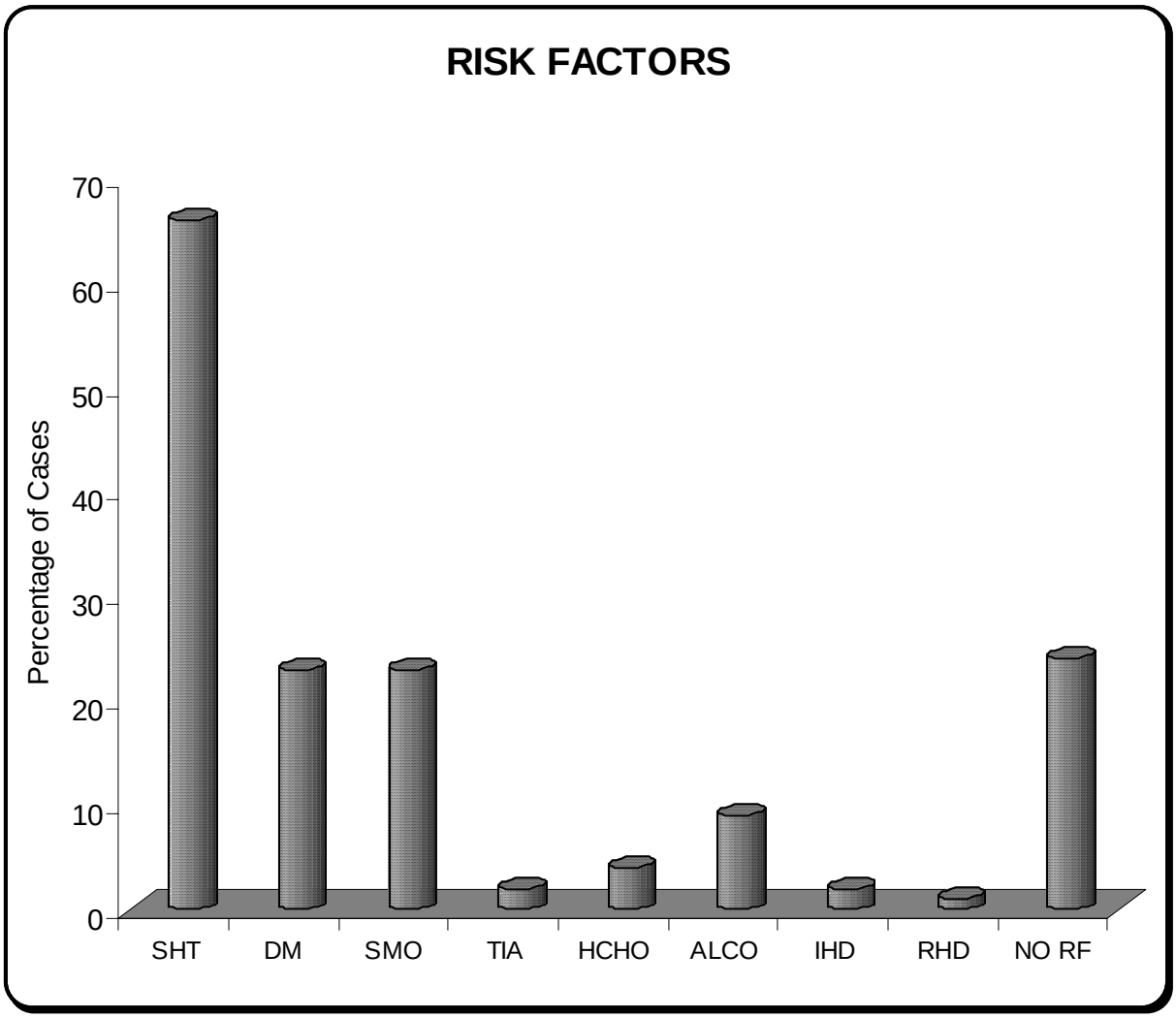
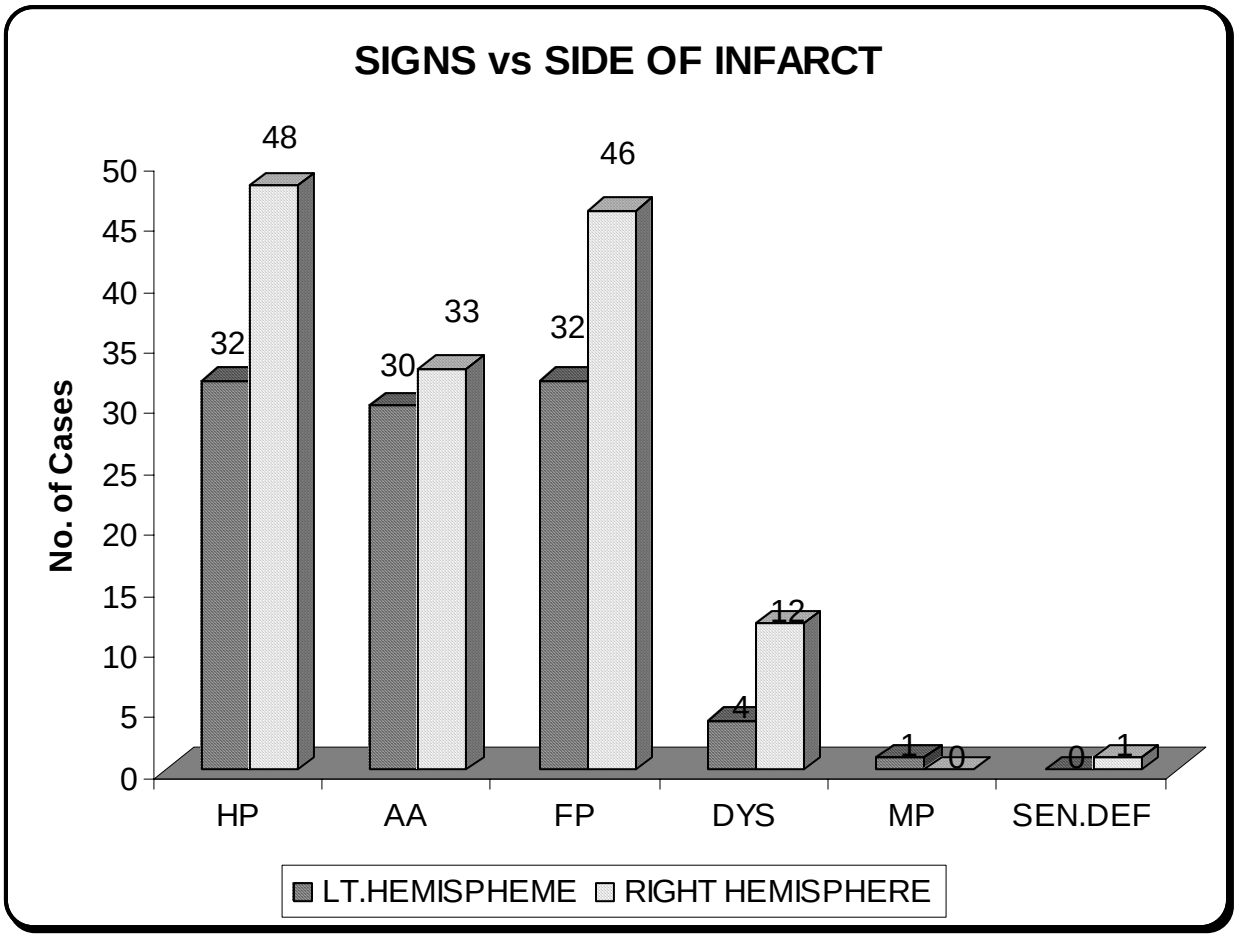
-



SHT – ***SYSTEMIC HYPERTENSION***
DM – ***DIABETES MELLITUS***
SMO – ***SMOKING***
TIA – ***TRANSIENT ISCHEMIC ATTACKS***
HCHO – ***HIGH CHOLESTEROL***
ALCO – ***ALCOHOLISM***
IHD – ***ISCHEMIC HEART DISEASE***
RHD – ***RHEUMATIC HEART DISEASE.***
NO RF - NO RISK FACTORS

-



SEN. DEF – SENSORY *DEFICITS*

DYS -DYSARTHRIA

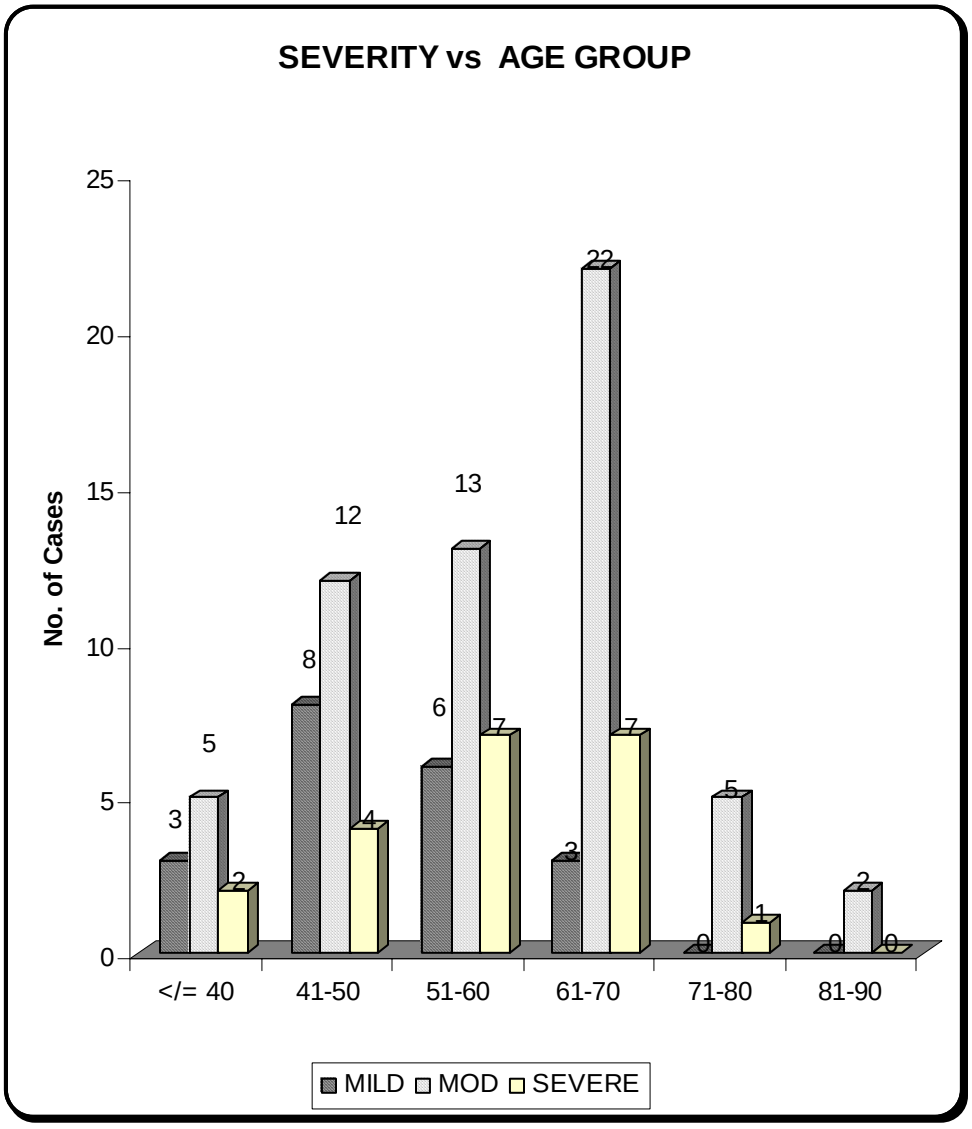
FP – *FACIAL PARESIS*

AA - APHASIA

HP – HEMIPARESIS/ HEMIPLEGIA

MP - MONOPARESIS

-



MILD STROKE – NIHSS SCORE <6

MODERATE STROKE –NIHSS SCORE 6-14

SEVERE STROKE – NIHSS SCORE >14

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BIBILIOGRAPHY

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PROFORMA

-

PROFORMA

I) <u>DEMOGRAPHICS</u> **DATE:**

SERIAL NO **IP. NO.**

NAME **AGE** **SEX**

ADDRESS

OCCUPATION **INCOME**

II) <u>STROKE PATHOLOGY</u>

DATE AND TIME OF STROKE

DATE OF ADMISSION

REASON FOR DELAY

ACTIVITY AT THE TIME OF STROKE ONSET

MODE OF ONSET AND STROKE BEHAVIOUR: 1)SUDDEN WITH MAXIMAL DEFICIT/

-

2) PROGRESSIVE/

3)FLUCTUATING/

4) REMiSSION/

STROKE TYPE : ISH/HGE/UNCERTAIN

LOCATION ; 1) RT. HEMISPHERE/

2) LT.HEMISPHERE/

3) BILATERAL/

4) BRAIN STEM

5) CEREBELLAR/

6) OTHERS.

METHOD OF STROKE DIAGNOSIS ; CLINICAL CRITERIA/CT/MRI

CT SCAN FINDING

HAEMORRHAGE Y/N

INFARCT Y/N

LOCATION AC/MC/PC

WATER SHED/GLOBAL/LACUNAR/OTHERS

AGE ACUTE/SUB ACUTE/OLD

TIME BETWEEN STROKE ON SET AND CT BRAIN :

PAST HISTORY OF STROKE : Y/N

ANY PERSISTING NEUROLOGICAL ILLNESS : Y/N

RANKIN SCALE (DOA) : 00/01/02/03/04/05

-

III)STROKE RIST FACTORS

SHT Y/N

DM Y/N

SMOKING Y/N

ALCOHOLISM Y/N

HIGH CHOLESSTEROL Y/N

IHD Y/N

RHD Y/N

AF Y/N

CAROTID STENOSIS Y/N

DISCHARGE STATUS : DHI /DHA /AH /EXP

DISCHARGE DATE

DURATION OF HOSPITALISATION

IV) ACUTE STROKE SYMPTOMS AND SIGNS

SYMPTOMS

HEADACHE

VERTIGO

VOMITING

GAIT DISTURBANCE

CONVULSIONS

SPEECH DEFICIT

-

SIGNS

SPEECH DEFICIT

HEMIANOPIA

DIPLOPIA

MOTOR SYSTEM

PARESIS AT ANY SITE

PARESIS OF ARMS **Y/N** **R/L/B**

PARESI OF LEGS **Y/N** **R/L/B**

PARESIS OF FACE **Y/N** **R/L/B**

FIRST INVOLVED **FACE / ARMS / LEGS**

NO SUCH ORDER **Y/N**

SENSORY DEFICIT **Y/N**

CEREBELLAR SIGNS **Y/N**

-

THE NATIONAL INSTITUTES OF HEALTH STROKE SCALE (NIHSS)
SCORE SHEET

ITEM	RESPONSE	SCORE
1a. Level of consciousness	**0 = Alert**	
	1 = Not alert	
	2 = Obtunded	
	3 = Unresponsive	
1b. Level of consciousness questions	**0 = Answers both correctly**	
	1 = Answers one correctly	
	2 =Answers neither correctly	
1c. Level of consciousness commands (2)		
	0 = Performs both tasks correctly	
	1 = Performs one task correctly	
	2 = Performs neither task	
2. Gaze		
	0 = Normal	
	1 = Partial gaze palsy	
	2 = Total gaze palsy	
3. Visual fields		
	0 = No visual loss	
	1 = Partial hemianopsia	
	2 = Complete hemianopsia	
	3 = Bilateral hemianopsia	

-

4. Facial palsy

0 = Normal

1 = Minor paralysis

2 = Partial paralysis

3 = Complete paralysis

5. Motor arm

a. Left

b. Right

0 = No drift

1 = Drift before 5 s

2 = Falls before 10 s

3 = No effort against gravity

4 = No movement

R **L**

6. Motor leg

a. Left

b. Right

0 = No drift

1 = Drift before 5 s

2 = Falls before 5 s

3 = No effort against gravity

4 = No movement

R **L**

-

7. Ataxia

0 = Absent

1 = One limb

2 = Two limbs

8. Sensory

0 = Normal

1 = Mild loss

2 = Severe loss

9. Language

0 = Normal

1 = Mild aphasia

2 = Severe aphasia

3 = Mute or global aphasia

10. Dysarthria

0 = Normal

1 = Mild

2 = Severe

11. Extinction / inattention

0 = Normal

1 = Mild

2 = Severe

DATE OF ADMISSION NIHSS SCORE →

DATE OF DISCHARGE NIHSS SCORE →

-

MASTER CHART

-

	RISK FACTORS	SYMPTOMS	SIGNS	NIHSS DOA	NIHSS DOD	DOH.	CT-FINDING	DI OI
	DM	ALC,GD	HP,FP	6	4	6	AC.RT. MC.LAC. INF	NI
	SHT,DM	GD,SD	HP,FP	7	7	6	AC.RT.MC INF	NI
	SHT	ALC,GD	COMATOSE	26	31	3	AC.LT.AC/MC/PC HGE	CT
	_	ALC,GD,SD	HP	14	0	2	**MULTIPLE GRANULOMA**	CT
	SHT,SMO	GD	HP,FP	5	2	2	AC.RT.MC.INF	NI
	SHT	ALC,HD	-	5	5	5	**LT FRONTAL MENINGIOMA**	CT
	SHT	ALC,SD	HP,FP	10	2	12	AC. RT.MC INF	NI
	SHT	ALC,HD,SD	HP,FP	10	3	5	AC.RT.MC INF	NI
	SHT,DM,FH	ALC,SD	HP,FP	22	28	1	AC.RT.MC INF(MASSIVE)	NI
	SHT	GD,SD	HP,FP	3	1	3	AC.LT.MC.LAC. INF	NI
	SHT	ALC,HD,SD	HP,FP	16	5	10	AC.RT.MC INF	NI
	SHT	ALC	HP,FP	11	6	5	AC.LT.MC INF	NI
	SHT,DM,SMO	ALC	HP,FP	18	30	3	AC.LT.MC HGE	CT
	SHT,DM	ALC	HP,FP	8	5	2	AC.LT.MC INF	NI
		GD,HD	HP,FP	5	0	2	**NORMAL**	**BI HY**
	SHT,SMO	ALC	HP,FP	13	5	6	AC.RT.MC.HGE	CT
	SHT	ALC,HD,VOM,CONV	COMATOSE	28	31	1	AC.RT.MC.HGE	CT
	SHT,SMO	ALC,GD	HP,FP	12	3	6	AC.RT.MC.INF	NI
	SHT,SMO	ALC,GD	HP,FP	9	4	4	AC.LT.MC.INF	NI
	_	GD	HP,FP	10	3	4	AC.RT.MC.INF	NI
	SHT	GD	HP,FP	4	4	10	AC.RT.MC.LAC.INF	NI
	SHT,SMO	ALC,GD,SD	HP,FP,SENSORY DEFICIT	12	4	7	AC.RT.MC.INF	NI
	SHT,DM,SMO	ALC,GD,SD	HP,FP	8	3	6	AC.LT.MC.INF	NI
	SHT	ALC,GD,SD	COMATOSE	26	30	1	AC..LT.MC.INF(MASSIVE)	NI
		ALC,GD,HD	HP	5	2	4	AC.RT.AC/MC LAC INF	NI
	SHT	ALC,GD,SD	HP,FP	6	4	4	AC.LT.MC.INF	NI
		ALC,GD,SD	HP,FP	9	4	5	AC.LT.MC.INF	NI
	SMO	ALC,GD,SD	HP,FP	8	3	2	AC.RT.MC.INF	NI
		GD,SD	HP,FP	4	3	7	**NORMAL**	NI
	SMO,CHO	ALC,GD,SD	HP,FP	9	4	17	AC.LT.MC.INF	NI
	SHT,DM,SMO,CHO	ALC,GD,SD	HP,FP	6	5	8	AC.RT.AC.INF	NI
	SHT	GD,SD	HP,FP	5	4	11	AC.RT.MC.LAC.INF	NI
		GD,SD	HP,FP	8	5	6	AC.LT.MC.INF	NI
	SMO	HD,SD	MP,FP	3	1	3	AC.LT.AC.INF	NI
	SHT	ALCGD,SD	HP,FP	22	6	10	AC.RT.MC.INF(MASSIVE)	NI
	SHT,DM	ALC,GDS,SD	HP,FP	9	5	10	AC.LT.MC.INF	NI
		GD	HP,FP	3	3	3	AC.RT.MC.INF	NI
	SHT,ALCOHOL	ALC,GD,SD	HP,FP	10	4	6	AC.LT.MC.INF	NI
		ALC,GD,SD	HP,FP	12	6	2	AC.RT.MC.INF	NI
	SHT,DM,SMO	ALC,GD,SD	HP	6	4	2	AC.RT.MC.LAC.INF	NI

		SD	HP	5	1	5	AC.LT.AC.INF	NI
	SHT,DM,SMO	ALC,GD,SD	HP,FP	13	5	4	AC.RT.MC.LAC.INF	NI
	SHT,DM,SMO	ALC,GD,HD,SD	HP,FP	30	11	5	AC.B/L MC.INF	NI
	SHT.DM	ALC,GD,SD	HP,FP	7	2	6	AC.RT.MC.HGE	CT
	SHT	ALC,GD,SD	HP,FP	6	4	3	AC.RT.MC.INF	NI
	SHT,CHO,ALCOHOL	ALC,GD,SD	HP,FP	7	3	4	AC.LT.MC.INF	NI
		ALC,GD,SD	HP,FP	18	5	7	AC.RT.MC.INF(MASSIVE)	NI
	SHT,DM	ALC,GD,SD	HP,FP	30	6	10	AC.LT.MC.INF	NI
		ALC,GD,SD	HP,FP	11	6	5	AC.LT.MC.INF	NI
	SHT	ALC,GD,SD	HP,FP	11	9	3	AC.RT.MC.INF	NI
		GD	HP,FP	3	1	8	AC.RT.MC.INF	NI
		GD	HP,FP	5	3	8	AC.RT.MC.INF	NI
	SHT	ALC,GD,SD	HP,FP	10	5	3	AC.LT.MC.INF	NI
	SHT	GD	HP,FP	5	5	11	AC.RT.MC.LAC.INF	NI
	SHT,DM,ALCOHOL	GD	HP,FP	7	6	5	AC.RT.MC.INF	NI
		GD	HP,FP	5	1	6	**NORMAL**	NI
		GD,SD	HP,FP	7	4	3	AC.RT.MC.INF	NI
	SHT,DM	ALC,GD,SD	HP,FP	10	5	9	AC.RT.PC.INF	NI
		GD,SD	HP,FP	12	5	7	AC.RT.MC.INF	NI
	SHT,DM,TIA	ALC,GD,SD	HP,FP	23	9	3	AC.LT.MC.INF	NI
	SHT	ALC,GD,SD	HP,FP	11	4	10	AC.LT.AC.INF	NI
	SHT	ALC,GD,SD	HP,FP	10	4	9	AC.RT.MC.INF	NI
	SMO	ALC,GD,SD	HP,FP	6	2	6	**NORMAL**	NI
	SHT,SMO	GD	HP,FP	4	3	4	AC.RT.MC.INF	NI
		ALC,HD,GD,SD		28	13	7	**SOL LT.FRONTAL REGION**	CT
	SMO,ALCOHOL	ALC,SD	HP,FP	8	5	4	AC.LT.AC.INF	NI
	SHT,ALCOHOL	ALC,GD,SD	HP,FP	10	5	13	AC.RT.MC.INF	NI
	SHT	GD,SD	HP,FP	6	3	6	AC.LT.MC.INF	NI
	SHT,SMO	ALC,GD,SD	HP,FP	7	1	3	AC.LT.MC.INF	NI
	SHT,DM,CHO	,ALC,GD,SD	HP,FP	18	8	5	AC.RT.MC.INF	NI
	SHT	ALC,GD,SD	HP	15	10	6	AC.LT.MC.INF	NI
	SMO,ALCOHOL	ALC,GD,SD	HP,FP	26	10	8	AC.LT.MC.INF(MASSIVE)	NI
	SHT,DM	GD	HP	4	2	11	AC.LT.MC.LAC.INF	NI
		GD	HP,FP	6	3	8	AC.RT.MC.INF	NI
	SHT	ALC,GD	HP,FP	10	3	2	AC.RT.MC.INF	NI
	SHT,DM	ALC,GD,SD	HP,FP	9	3	3	AC.LT.MC.INF	NI
	DM	ALC,GD,SD	HP,FP	9	3	4	AC.RT,MC.INF	NI
		GD	HP,FP	8	3	6	AC.RT.AC.INF	NI
	SHT	ALC,VOM,CONV,SD	COMATOSE	24	30	2	AC.RT.MC.INF(MASSIVE)	NI
	SHT	ALC,GD	HP,FP	7	3	3	AC.RT.MC.INF	NI
		ALC,GD	HP,FP	14	3	2	AC.LT.MC.INF	NI
	SHT	ALC,GD	COMATOSE	24	28	1	AC.BR.STEM.INF	CT
	DM,SMO	ALC,GD	HP,FP	10	3	8	AC.RT.MC.INF	NI
	SHT	GD	HP,FP	5	3	12	**NORMAL**	NI
	SMO,ALCOHOL	ALC,GD	HP,FP	8	4	4	AC.RT.MC.INF	NI
		ALC,GD,SD	HP,FP	10	4	19	AC.LT.MC.INF	NI
	SHT	GD	HD,FP	14	3	3	AC.RT.MC.LAC.INF	NI
	SHT	ALC,GD,SD	HP,FP	9	4	7	AC.LT.MC.INF	NI

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		ALC,GD,SD	COMATOSE	23	32	5	AC.RT.MC.INF	CT
	SHT,DM	ALC,GD,SD	HP,FP	18	3	10	AC.RT.MC.INF(MASSIVE)	NI
		ALC,GD,SD	HP,FP	7	2	6	AC.RT.MC.INF	NI
	SHT,FH	GD	HP,FP	6	3	9	AC.RT.MC.INF	NI
		GD	HP,FP	8	5	6	AC.LT.MC.INF	NI
	SHT	GD	HP,FP	3	3	6	**NORMAL**	NI
	SHT	ALC,GD,SD	HP,FP	8	4	4	AC.LT.MC.INF	NI
	SMO	ALC,GD,SD	HP,FP	7	3	4	AC.RT.AC.INF	NI
	TIA	GD	HP,FP,HEMIANOPIA	10	5	27	AC.RT.MC.INF	NI
	SHT	ALC,GD,SD	HP,FP	6	3	8	AC.RT.MC.LAC.INF	NI
	SHT,SMO	ALC,GD,SD	HP,FP	18	5	11	AC.LT.MC.HGE	CT
	SHT,DM	ALC,GD,CONV,SD	HP,FP	26	6	6	AC.LT.MC.HGE	CT

A-ACTIVITY
AC-ANTERIOR CEREBRAL ARTERY
AC-ACUTE
AH-ACUTE HOSPITAL
ALC-ALTERED LEVEL OF CONSCIOUSNESS
CHO-HIGH CHOLESTEROL
CONV-CONVULSIONS
DHA- DISCHARGED WITH HOME ASSISTANCE
DHI-DISCHARGED WITH HOME INDEPENDENCE
DM-DIABETES MELLITUS
DOHS-DURATION OF HOSPITAL STAY
EXP-EXPIRED
FH-FAMILY HISTORY
FP-FACIAL PARESIS
GD-GAIT DISTURBANCE
HD-HEADACHE
HP-HEMIPARESIS/HEMIPLEGIA
HGE-HEMORRHAGE
INF-INFARCT
LAC-LACUNAR
LT-LEFT
MC-MIDDLE CEREBRAL ARTERY
MP-MONOPARESIS
NIHSS-NATIONAL INSTITUTES OF HEALTH STROKE SCALE
PC-POSTERIOR CEREBRAL ARTERY RT-RIGHT
S-SLEEP,
SD-SPEECH DEFICIT
SHT-SYSTEMIC HYPERTENSION
SMO-SMOKING
SOL-SPACE OCCUPYING LESION
TOC-TIME OF OCCURENCE
VOM-VOMITING
TIA-TRANSIENT ISCHEMIC ATTACK